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OTOSCLEROSIS IN IDENTICAL TWINS

RESULT OF FENESTRATION

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Since the publication of Lempert's description of his fenestration operation[1] and the impetus it gave to aural surgeons throughout the world, thousands of hopeful otosclerotics have presented themselves for examination and possible surgery.

In clinical practice and autopsy, otosclerosis is a fairly common finding[2]. In identical twins, however, otosclerosis appears to be rarely encountered. The literature does not reveal more than a score of such cases.

Fowler[3] reported his findings in three pairs of identical twins with otosclerosis and reviewed the histories of six pairs reported by others. Since then, he repeatedly tested many of 150 twins in the public schools of New York City. No progressive deafness or positive family history was discovered. In 1947 he reported a further five cases of otosclerosis in identical twins[4].

The following were his salient observations:—

i. The mother's milk was deficient in every instance.

ii. In the eight females, menstruation began earlier in the twin who is now the deafer in each pair.

iii. During several years, little if any increase in deafness has been noted in these twins, in spite of several childbirths and upper respiratory infections in several instances.

iv. With the exception of one individual, the deafness was shared by both individuals in each pair and by both ears of both individuals in each pair. This is interpreted as indicating that in these twins, either the hereditary or environmental factors, or both, must have had similar etiological effects in every pair but one.

The identical twins here presented are females aged 23 years. They were first seen in January 1948. Both had been wearing a hearing aid in the left (better) ear for four and a half years. The similarities and differences will be seen from the accompanying table. 'A' is the firstborn and the slightly deafer clinically.

SIMILARITIES AND DIFFERENCES

	A	*B*
Age:	23	23
Sex:	Female	Female
Occupation:	Typiste	Typiste
Family History of Deafness:	Pos.	Pos.
Weight at Birth:	4 lbs.	4½ lbs.
Menses at:	13	13½
Age Deafness first noted:	13	15
Tinnitus:	Yes	Yes
Exanthemata:	Same	Same
Colds:	Same	Same
Earache or Discharge:	No	No
Lymphoid Tissue in Nasopharynx:	More	Little
Allergies:	Nil	Nil

Finger Prints: These were taken and compared with this conclusion: 'Their finger prints are found to be absolutely unlike in the characteristics that are used for establishing identity. A general likeness in patterns is evident, but the resemblance is only of interest because it is known that the two ladies are twins'.

The following examinations and tests were performed prior to fenestration:—

i. *Clinical*: (*a*) Wassermann test. (*b*) X-ray of mastoids. (*c*) Patency of eustachian tubes. (*d*) Direct nasopharyngoscopic examination. (*e*) Membrana tympani.

ii. *Audiograms repeated at intervals*: (*a*) Air conduction. (*b*) Bone conduction with masking. The bone conduction loss in the speech frequencies was small, indicating a good cochlear reserve.

iii. *Tuning-fork tests*: 512, 1024, 2048. Tuning-forks were tested on the mastoids with masking.

iv. *Speech testing*: (*a*) Whispered voice; (*b*) conversation voice; (*c*) intelligibility for speech: (1) By voice; (2) by recorded speech; (3) by microphone.

v. *Assessment*: The twins were assessed as to suitability for fenestration by the Otosclerotic Board functioning at the Hospital.

Fenestration: The operation was performed according to the technique of Lempert, the endaural approach being used in each case. The anaesthesia too, was similar to that employed by Lempert at his clinic, a

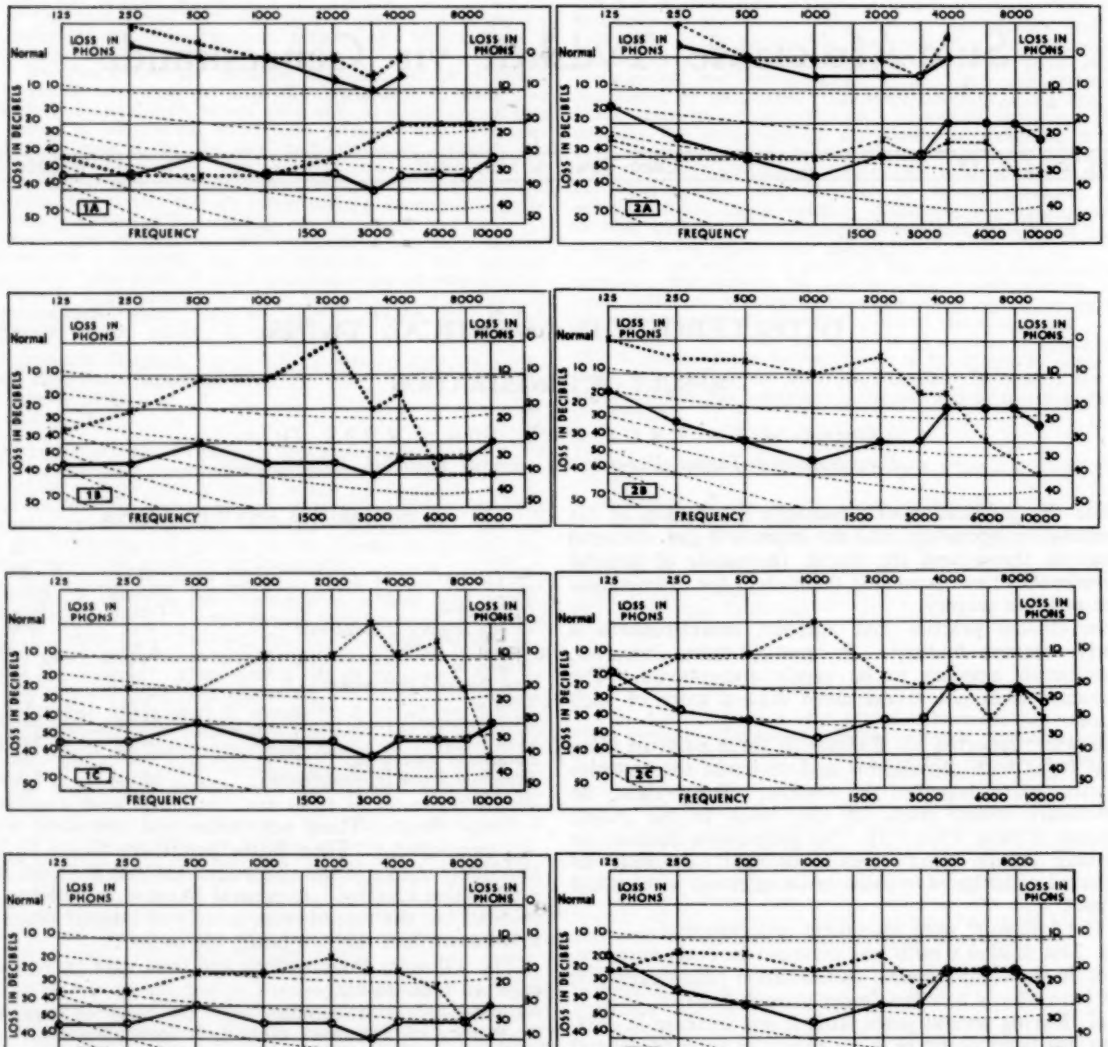

Fig. 1 (a). Twin A (Pre-Operative).
Air { O = Right Ear; X = Left Ear } Bone { ▶ = Right Ear; ◀ = Left Ear }

Fig. 1 (b). Twin A (Three Weeks Post-Operative).
O = Pre-Operative
X = Post-Operative

Fig. 1 (c). Twin A (Three Months Post-Operative).
O = Pre-Operative
X = Post-Operative

Fig. 1 (d). Twin A (Six Months Post-Operative)
O = Pre-Operative
X = Post-Operative

Fig. 2 (a). Twin B (Pre-Operative)
Air { O = Right Ear; X = Left Ear } Bone { ▷ = Right Ear; ◁ = Left Ear }

Fig. 2 (b). Twin B (Three Weeks Post-Operative)
O = Pre-Operative
X = Post-Operative

Fig. 2 (c). Twin B (Three Months Post-Operative)
O = Pre-Operative
X = Post-Operative

Fig. 2 (d). Twin B (Six Months Post-Operative)
O = Pre-Operative
X = Post-Operative

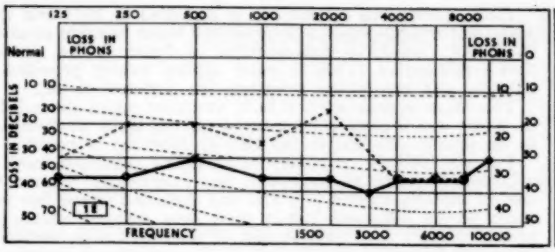

Fig. 1 (e). Twin A (12 Months Post-Operative)
O = Pre-Operative
X = Post-Operative

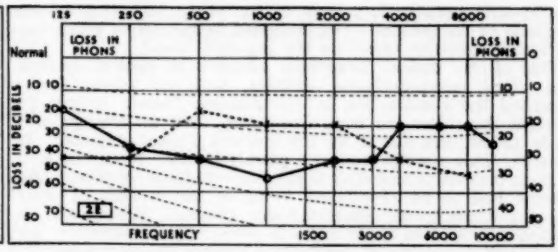

Fig. 2 (e). Twin B (12 Months Post-Operative)
O = Pre-Operative
X = Post-Operative

mixture of local infiltration plus sedation with barbiturates.

'A' was fenestrated on 18 August 1948 and 'B' on 25 August 1948. Fenestration in both cases was performed on the right ear, which is the deafer ear in both individuals.

Audiograms: The audiograms were taken (1) preoperatively; (2) three weeks post-operatively; (3) three months post-operatively; (4) six months post-operatively; (5) twelve months post-operatively.

Result: Although the audiograms do not show a dramatic gain in decibels, the speech frequencies are 10 decibels above the practical 30, decibel level. Pure tone audiometry significantly underestimates the gain for speech[5]. Their ability to understand speech, person to person, is excellent, and in group conversation, good.

SUMMARY

1. A case of otosclerosis in identical twins is reported.
2. The results of fenestration one year post-operatively are shown. 3. As far as is known from the literature, an otosclerotic twin has not previously been fenestrated.

I wish to express my thanks to Prof. P. de V. Pienaar of the Department of Phonetics and Logopedics of the University of Witwatersrand, and to Mr. W. van der Sandt, Clinical Audiologist, for the excellent audiograms and for their keen interest.

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MODERN METHODS OF MALARIA CONTROL*

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In a recent review of the last 50 years a leading English weekly records the great advance of medicine and surgery and features cures for malaria, blood transfusions at any time and in any place, the wonder cures and pain alleviations of penicillin and M. & B. and the insulin check on the scourge of diabetes. It quotes these as but forerunners of advances already in the process of design and development in the laboratories all over the world.

This review is strangely silent about the equally dramatic advances which have been made in the field of preventive medicine. This is perhaps symptomatic of the attitude of the average man and woman who lauds the treatment which relieves the pain and suffering of disease but is largely unaware of the measures taken by Governments and Local Authorities to prevent outbreaks of disease. Probably in no other disease which the human flesh is heir to has the institution of control measures yielded more spectacular results than in the case of malaria. We need look no further than our own country for evidence of this. Vital statistics reveal that during 1932 approximately 10,000 deaths occurred in Natal and Zululand from malaria and in the two Government Hospitals in Durban alone 2,252 cases suffering from this disease were admitted. To-day it is exceedingly rare for a death to be reported from malaria and medical students complain of the lack of cases for study in our hospitals.

To appreciate fully what has been achieved in the field of preventing malaria it is necessary briefly to sketch the history of this disease.

As its name implies, the disease was originally attributed to the miasmas or bad airs arising from swamps and low-lying areas, hence malaria meaning literally bad airs.

Research workers early in this century established the fact that malaria was due to a parasite which destroyed the red blood cells and which was transmitted to human beings by the females of the Anopheline group of mosquito. These mosquitoes became infected after biting humans who harbour the parasites in their blood.

Perhaps we should not be too harsh in condemning the female mosquito. She, after all, is only concerned with providing food for her unborn young. The blood she sucks up is stored as nutriment in her eggs. Unfortunately she injects her saliva before she sucks our blood and, if she is infected,

* Radio Talk by Dr. F. W. P. Cluver, Deputy Chief Health Officer, Union Health Department, broadcast from the Durban Studio of the South African Broadcasting Corporation.

the malaria parasite is present in her saliva. Incidentally the itchiness of the mosquito bite is caused by this saliva which has an anti-coagulative effect on the ingested blood.

One of the earliest large-scale attempts to control malaria and other mosquito-borne diseases, such as yellow fever, preceded the construction of the Panama Canal. It will be recalled that after the Suez Canal was built an attempt was made to cut this Canal by the eminent French engineer, de Lesseps. This attempt proved a ghastly failure. Workmen died in their thousands and after expending vast sums of money the scheme had to be abandoned owing to the ravages of what we now know to be mosquito-borne diseases. When, later, a second attempt was made research workers had established the causes of the diseases which had ruined the first attempt. Health workers embarked on anti-mosquito measures before the commencement of construction. The methods employed were mainly directed against the larval stage by spraying kerosene on breeding pools. Kerosene or paraffin forms a film on the surface of the water and thus suffocates the larval mosquito which is a surface breather. The draining of stagnant pools and the provision of gauze-screened houses for the employees enabled the workers to ward off malaria and yellow fever and the work was carried on to a triumphant conclusion.

These early methods of control were necessarily costly and only applicable in limited areas.

It had long been known that the taking of quinine regularly would ward off attacks of the disease. The introduction of drugs such as Atebrin in 1930 (and later Paludrin) was successfully exploited during the last war when Japan early in 1942 had seized the world's main sources of quinine. By regularly taking these suppressive drugs troops were able to operate in intensely malarious areas. The taking of these suppressive drugs did not prevent infection but did enable troops to carry on with their jobs by restraining the parasites in their systems from working their fell purpose.

The Union Government realising that some of the most productive parts of the country were largely undeveloped owing to the scourge of malaria invited Professor Swellengrebel, a noted malariologist, to investigate the problem. Professor Swellengrebel's report published in 1931 emphasized that our malaria incidence was due to two species of Anopheles—*gambia* and *funestus*—and advised species sanitation to be directed against these two vectors. It was soon found that to employ the older anti-larval methods against these vectors would be largely impracticable if the measures were to be applied in rural areas and native reserves which constituted the main reservoirs of infection.

Our main malaria vector in the Union—*Anopheles gambiae*—which transmits the disease in epidemic form, breeds most prolifically in small exposed devegetated pools formed by cattle hoof-marks, wheel tracks, etc. It was found to be a practical impossibility to oil efficiently or to employ other anti-larval methods in native areas. The Bantu is essentially a pastorialist and cattle have not only an economic but also a ritualistic significance and any interference with grazing would have been strongly resented.

The fact that the vectors of the disease in the Union were intensely house-haunting in their habits led to the inauguration of house-spraying with insecticide. It was found that the spraying of dwellings weekly or better still twice weekly with Pyrethrum dissolved in paraffin effectively killed the harbouring mosquitoes. An insecticidal campaign was embarked on in Natal and Zululand and later in the Transvaal Lowveld. The beneficial results were soon apparent. Malaria admissions in the two Government hospitals in Durban dropped from 2,252 in 1932 to 68 in 1936.

The pyrethrum insecticidal spraying of dwellings, however, merely destroyed the mosquitoes actually harbouring in the houses.

The introduction of D.D.T. which had been successfully employed by the military forces towards the end of the war, particularly against typhus in Naples, forged a new weapon against malaria.

It was found that when the walls of dwellings were sprayed with D.D.T. dissolved in paraffin a film remained which destroyed mosquitoes resting on such walls. Moreover, the effect lasted for at least three months. Every house sprayed with D.D.T. thus became a lethal chamber to entering mosquitoes but had no deleterious effect on the human occupants.

Field experiments carried out in Zululand in 1947 and 1948 with various dilutions of a concentrated water-miscible emulsion of D.D.T., which is manufactured in South Africa, showed that a dilution of 1:300 in water applied weekly provided an effective and economical substitute for anti-malaria oil.

The same concentrated D.D.T. emulsion when diluted 1:8 in water was found, when sprayed on the walls of dwellings, to be equally effective to the D.D.T. dissolved in paraffin.

The employment of concentrated D.D.T. emulsion both as a larvicide and as an insecticide has enormously reduced the cost of malaria control in Natal and Zululand where last year some 92,476 huts and rooms were thus sprayed effecting a saving of more than 90% in cost of material and in transport. In round figures it may be stated that spraying a room with D.D.T. emulsion, thereby ensuring the destruction of malaria mosquitoes in it for at least three months actually costs only 9d.

The World Health Organization meeting in Rome last year emphasized the need for international malaria control on a worldwide basis. While each year malaria in one area after another is retreating dramatically as a result of such new insecticides as D.D.T. yet the disease still has high incidence in large areas causing hundreds of millions of cases and millions of deaths. Malaria still holds back, or makes impossible, food production in undeveloped areas; it still interferes seriously with industrial and agricultural activities in much of the tropics and sub-tropics; and it still takes a high toll of victims in infancy and early childhood. The fact was stressed that by virtue of insecticides developed since 1940 it is now possible to obtain a degree of malaria control amounting in some cases to actual eradication. During the past year, the report continues, numerous additional examples of the successful application of residual insecticides have abundantly confirmed this fact. Malaria is now highly vulnerable to planned attack and the ultimate aim of the World Health Organization should be to eliminate this disease as a public health problem from the world.

In certain islands in the Mediterranean such as Sardinia and Cyprus where malaria has been endemic from time immemorial, campaigns are now in progress which aim not only at controlling malaria but the total eradication of the insect vectors. The initial cost of such a campaign involving as it does both anti-larval and anti-adult destruction of the vectors is high but, once eliminated, the cost of annual control is done away with, although a sentinel service will have to be maintained to guard against the re-infestation of the island by transported mosquitoes. It is well known that mosquitoes from the west coast of Africa have been carried across the Atlantic to Brazil both in ships and aircraft. In fact Brazil experienced one of its worst outbreaks of malaria following the introduction of *Anopheles gambiae* from West Africa and a large-scale and costly campaign was necessary before this introduced mosquito was eradicated. In addition to the control methods referred to there are certain permanent measures which are often very effective in eliminating breeding foci of malaria vectors.

Travellers in Zululand sometimes comment on the many clumps of eucalyptus or gum trees which dot the low-lying areas. It is probably not realized that these odd gum plantations were at one time heavy vector breeding areas. The planting of these gum trees, which are very greedy drinkers, has dried up the seepages which formerly occurred and served to breed malarial mosquitoes. Though they were deliberately planted as a long-range preventive measure they now constitute a valuable economic asset.

Pessimists have dolefully pointed out that the world's increasing population is outgrowing its food production. It has even been stated that disease prevention means more malnutrition if not actual starvation by removing the so-called natural checks on population increase. Whatever force these arguments may have does certainly not apply in the case of malaria and other tropical diseases.

The tropics and subtropics could, if systematically developed, enormously increase the world's food production. Most of these areas are fertile and well watered and are not productive owing to the ravages of these tropical diseases. There are vast areas of tropical Africa where cereal crops can be harvested twice a year from the same fields whereas temperate climates produce only one annual crop.

The conquest of tropical diseases will free vast areas in the tropics and subtropics for food production which should adequately meet every forseeable requirement of the future.

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VAN DIE REDAKSIE

EDITORIAL

PHARMACOPOEA INTERNATIONALIS

Die eerste proewe van die *Pharmacopoea Internationalis* was in die hande van die Deskundige Komitee oor die Samevoeging van Farmakopeias gedurende sy vyfde sessie wat van 26 September tot 5 Oktober 1949 te Genève gehou is. Hierdie vergadering was die laaste stap voordat die eerste uitgawe van hierdie werk uitgegee word, wat volgens voorneme aan die begin van 1950 sal gebeur.

Die monografieë, algemene kennisgewings en aanhangsels is voltooi en in hulle finale vorm opgestel. Die struktuurformules van die stowwe wat in die Farmakopeia opgeneem sal word, is volgens die stelsel van Patterson opgestel wat dit in 1940 in samewerking met Capell in 'n boek, *The Ring Index: Ring Systems Used in Organic Chemistry* uitgegee het. In hierdie werk het Patterson 'n stelsel probeer uitwerk om sikliese verbindinge te nommer en te benoem deur middel waarvan 'n mate van orde in die naamgewing van organiese verbindings geskep kan word, wat tans van land tot land verskil. Die skeikundige name van die stowwe in die Farmakopeia—beide in Engels en in Frans—is gekies om te voldoen aan die lys van radikale wat deur die Internasionale Skeikundige Unie gepubliseer is.

Gedurende die ondersoek van die monografieë oor antitoksiese serums en sekere plantaardige geneesmiddels is die vraag oorweeg of die metode van biologiese kragtigheidsbepaling vir al hierdie stowwe ingesluit moet word. In baie lande is daar regulasies wat die toetse bepaal waaraan sulke stowwe moet voldoen en die metodes omskryf waarvolgens die toetse uitgevoer moet word. Dit mag dus onnodig voorkom om metodes van biologiese kragtigheidsbepaling in die *Pharmacopoea Internationalis* te beskryf. Die Komitee se besprekings het egter aan die lig gebring dat verskeie lande geen amptelike metode het om die kragtigheid van die betrokke stowwe te bepaal nie en het aangetoon dat die *Pharmacopoea Internationalis* deur sommige standaardmetodes te publiseer, nuttige inligting aan die owerhede wat regulasies wil opstel asook aan farmaseute en geneeshere kan verskaf. Dus sal die metodes wat tans as die bevredigendste beskou word in die *Pharmacopoea Internationalis* opgeneem word. Die keuse van hierdie metodes is in samewerking met die deskundige komitee oor Biologiese Normalisering gedoen. Dit hoef byna nie genoem te word dat hierdie metodes, asook skeikundige en fisiese metodes van kragtigheidsbepaling in latere uitgawes van die Farmakopeia verander mag word indien sodanige veranderings as gevolg van nuwe navorsing nodig word.

Die monografieë oor die toksoied van witseerkeel wat òf vir die Schicktoets òf as voorbehoedmiddel gebruik word en waarvoor daar nog geen internasionale stan-

The first proofs of the International Pharmacopoeia (*Pharmacopoea Internationalis*) were in the hands of the Expert Committee on the Unification of Pharmacopoeias during its fifth session, held in Geneva from 26 September to 5 October 1949. This meeting marked the last stage before publication of the first edition of this work, planned for the beginning of 1950.

The monographs, general notices and appendices were completed and put in their final form. The structural formulae of the substances which will be included in the Pharmacopoeia were drawn up according to the Patterson system, published in 1940, with the collaboration of Capell in a book entitled *The Ring Index: Ring Systems Used in Organic Chemistry*. In this work Patterson has tried to establish a system for numbering and naming cyclic compounds, by means of which some order could be brought into the nomenclature of organic compounds, which at present varies from country to country. The chemical names of the substances in the Pharmacopoeia—both in English and French—were chosen to conform with the list of radicals published by the International Union of Chemistry.

During the examination of the monographs on antitoxic sera and on certain vegetable drugs, the question was considered whether the method of biological assay should be included for all these substances. In many countries there are regulations stipulating the tests with which such substances should comply and defining the methods by which the tests should be carried out. It might therefore appear unnecessary to describe methods of biological assay in the International Pharmacopoeia. The committee's discussions revealed, however, that several countries have no official method of assay for the substances in question, and showed that the International Pharmacopoeia, by publishing some standard methods, could provide useful information for the authorities wishing to lay down regulations, as well as for pharmacists and doctors. Thus, the methods at present considered most satisfactory will be included in the International Pharmacopoeia. The choice of these methods was made in agreement with the Expert Committee on Biological Standardization. It need hardly be said that these methods, as well as chemical or physical methods of assay, may be modified in subsequent editions of the Pharmacopoeia, if such modification is made necessary by the results of fresh research.

The monographs dealing with diphtheria toxoid, which is used either for the Schick test or for prophylactic purposes, and for which no international standards

daarde bestaan nie, is vir publikasie in 'n aanhangsel of later uitgawe van die Farmakopeia uitgestel.

Die tafel van gebruiklike en maksimale dosisse van die geneesmiddels wat in die *Pharmacopoea Internationalis* voorkom, sal net dosisse vir volwassenes bevat. Die dosisse vir kinders sal later gepubliseer word. Daar is beklemtoon dat so 'n lyn wat gegrond is op ondervinding wat in verband met kindergeneeskunde opgedoen is, van belang sal wees aangesien dit in hierdie vorm slegs in 'n paar nasionale farmakopeias aangetref word.

Insluiting van Monografieë oor Nuwe Geneesmiddels. Sommige van die sintetiese antimalaria-geneesmiddels waarvan die doeltreffendheid bewys is, soos mepakien, pamakien en pentakien sal op aanbeveling van die Farmakopeia ingesluit word. Monografieë oor verskeie sintetiese plaasvervangers vir morfien wat deur die deskundige komitee oor Gewoontevormende Geneesmiddels genoem is, sal later gepubliseer word. Veral die volgende geneesmiddels is daarby betrokke: Amidone, dihidro-kodeïnon-bitartraat (geregistreerde naam: Dikodide), dihidro-oksikodeïnon-hidrokloried (geregistreerde naam: Eucodal) en metopon.

Nie-patent Name van Geneesmiddels. Gedurende sy vorige sessie het die Komitee dit beoog om internasionale nie-patente name aan belangrike nuwe geneesmiddels te gee wat op die internasionale mark geplaas word. Hierdie plan het lewendige belangstelling gewek. Die begeerte om die huidige verwarring op te los is in verskeie kringe en selfs deur sekere regerings uitgespreek en die Internasionale Farmaseutiese Federasie het reeds 'n voorlopige studie van die kwessie gemaak. Soos voorheen aangedui, is dit 'n probleem wat moeilikhede van wetlike aard oplewer en waarvoor geen onmiddellike oplossing verwag kan word nie. Dit is dus belangrik dat die Internasionale Unie vir die Beskerming van Nywerheidseiendom sowel as regerings geraadpleeg word en dat ook die ondersteuning van die nasionale farmakopiese kommissies en die koöperasies van vervaardigers van farmaseutiese produkte verkry word. Eers nadat die menings van hierdie verskillende liggame verkry is, sal die komitee in staat wees om aan die Uitvoerende Raad spesifieke voorstelle vir die ontwerp van 'n stelsel van nie-patente name voor te lê.

Lys van Sinonieme. Die insluiting van sinonieme in die *Pharmacopoea Internationalis* word nie beoog nie behalwe in uitsonderlike gevalle; daarbenewens sal nòg die geregistreerde name nòg die nie-patente name aangegee word wat in verskillende lande vir dieselfde middel gebruik word. Met die oog op toenemende moeilikhede wat oral teëgekom word en waarop veral die deskundige komitees van die Wêreldgesondheidsorganisasie wys, en wat veroorsaak word deur die veelvuldigheid van name vir dieselfde middel, het die Komitee op aanbeveling van die Deskundige Komitee oor Geestesgesondheid besluit om 'n lys van die vernaamste sinonieme (nie-patente name en geregistreerde name) vir geneesmiddels wat in die *Pharmacopoea Internationalis* voorkom benewens ander belangrike middels op die internasionale mark op te stel. Die voorneme bestaan om hierdie lys gedurende 1950 as 'n aanhangsel van die *Bulletin of the World Health Organisation* te publiseer.

as yet exist, were deferred for publication in a supplement or in a future edition of the Pharmacopoeia.

The table of usual and maximal doses of drugs figuring in the International Pharmacopoeia will include only doses for adults. The dosage for children will be published later. It has been stressed that such a list, founded on experience acquired in pediatrics, would be of interest, since it is found in this form in only a few national pharmacopoeias.

Inclusion of Monographs on New Drugs. Some of the synthetic anti-malarial drugs of proved efficacy, such as mepacrine, pamaquine and pentaquine, will be included in the first edition of the Pharmacopoeia, on the recommendation of the Expert Committee on Malaria. Monographs dealing with several synthetic substitutes for morphine, indicated by the Expert Committee on Habit-Forming Drugs, will be published later. The following products in particular are involved: amidone, dihydro-codeinone bitartrate (registered name: Dicodide), dihydro-oxycodeinone hydrochloride (registered name: Eucodal), and metopon.

Non-proprietary Names for Drugs. During its preceding session the committee had contemplated establishing international non-proprietary names for important new drugs placed on the international market. This proposal has aroused lively interest. The need for resolving the present confusion has been expressed in various quarters and even by certain governments, and the International Pharmaceutical Federation had already made a preliminary study of the question. As previously pointed out, this is a problem which raises difficulties of a legal nature, and for which no immediate solution can be expected. It is, therefore, important to consult the International Union for the Protection of Industrial Property, as well as governments and, further, to obtain the support of the national pharmacopoeial commissions and the co-operation of the manufacturers of pharmaceutical products. Only after having received the views of these various bodies will the committee be able to submit to the Executive Board specific proposals for establishing a system of non-proprietary names.

List of Synonyms. The inclusion of synonyms in the International Pharmacopoeia is not contemplated, except in very special cases; furthermore, neither registered names nor the non-proprietary names used in different countries for the same drug will be given. In view of the growing difficulties—encountered on all sides and pointed out, in particular, by the WHO expert committees—caused by the multiplicity of names for the same drug, the committee decided, on the recommendation of the Expert Committee on Mental Health, to prepare a list of the principal synonyms (non-proprietary names and registered names) for drugs appearing in the International Pharmacopoeia as well as for other important drugs on the international market. It is intended to publish this list, during 1950, as a supplement to the *Bulletin of the World Health Organization.*

ARGEMONE MEXICANA SEED POISONING

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On 29 August 1947, the District Surgeon of one of the North-Western Cape Districts wrote to the magistrate stating that there was an 'epidemic of a peculiar disease on Farm A, where there were seven coloured people suffering from the following symptoms: swelling of the legs; breathlessness; slight enlargement of liver; occasional diarrhoea.

Six members of one family and one member of another family were affected, and there has been one death'.

At that time the District Surgeon considered this was a nutritional disease. It was stated, however, that the farmer would not accept his diagnosis and in the farmer's opinion it was an infectious disease.

At the same time the District Surgeon stated that there had been a similar outbreak on Farm B in 1946 when five Coloured persons had been affected and three had died. He stated further that the onset in the 1946 outbreak was explosive and so was the onset in the present one. Both outbreaks had occurred just after the winter months.

The autopsy conducted on the person who had died in the 1947 outbreak showed no definite cause of death and the District Surgeon expressed his opinion that this matter was most urgent.

INVESTIGATION BY UNION HEALTH DEPARTMENT

The writer, at that time Assistant Health Officer, Union Health Department, was sent to investigate this outbreak, which was possibly an outbreak of senecio poisoning. This opinion was partly confirmed by the owner of Farm A, who had observed that fowls fed the same wheat that had been fed to his Coloured employees, had developed a similar condition. In addition the farmer stated that this wheat was contaminated with the seeds of what is known in that area as the *bloudissel*, i.e., *Argemone Mexicana* or Mexican poppy. He, therefore, attributed the outbreak to the seed of this plant which was present in the wheat and his conclusion was eventually found to be the correct one.

CLINICAL FINDINGS AND SIGNS

This condition is one which is fairly common in India and is described by Manson-Bahr and in Stitt's *Diagnosis, Prevention and Treatemnt of Tropical Diseases* (7th edition) *as Mustard Oil Poisoning* (*Epidemic Dropsy*).

At the time the writer could not obtain any literature on *Argemone Mexicana* seed poisoning locally, and did not know that mustard oil poisoning (epidemic dropsy) was the same condition.

CLINICAL FINDINGS: EASTERN COUNTRIES [1 2]

1. *Dropsy* is almost invariably present, usually appearing first in the legs and in some instances confined to them. In others it involves the entire body and clinically is very persistent, recurring during convalescence.

2. *Fever* is also very constant. Sometimes it precedes and sometimes it follows the dropsy. It is rarely high, usually ranging from 99° F. to 102° F.

3. *Diarrhoea and vomiting* generally ushered in the disease in an epidemic which occurred in Mauritius, while in cases which occurred in Calcutta these symptoms were not so frequent although by no means rare, and occurred at both the earlier and the later stages. The total duration is about six weeks.

4. *Peripheral neuritis* is absent and the knee jerks are present, but usually distressing aching of muscles, bones and joints, is prominent.

5. *Skin.* Exanthemata, erythematous on the face, rubeolar on the trunk and limbs, was frequently seen in Mauritius but less so in Calcutta. It appeared about a week after the oedema.

On the skin, vascular naevi often appear and bleed profusely, while telangiectases are common. Manson-Bahr states that Dee and Chatterjee described the eruptions as nodular in some cases. Lesions on the mucous membranes have been noted. Bleeding of these lesions may occur and may be uncontrollable. Ecchymotic patches consist not of haemorrhages but of telangiectases. In three to six weeks after the first symptoms nodular excrescences are seen. There may be 100 or more. They may be sessile or pedunculated, vary from the size of a pea to a lemon and may bleed readily.

6. *Cardio-Vascular System, etc.* Disturbances of the heart and circulation are prominent in nearly all the cases. The pulse is weak, rapid and irregular, the blood pressure low, cardiac bruits are often noted; breathlessness from exertion occurred in all cases and orthopnea in many. Signs of pleural or pericardial effusion, of oedema of the lungs, or pneumonia and cardiac dilatation are common. Hawes states that the lung signs are characteristic and resemble a bronchial spasm with defective aeration. Anaemia is usually marked and so are wasting and prostration.

7. *The Urine* is not albuminous, but of low specific gravity and greatly increased in amount.

8. *Glaucoma.* The commencement of glaucoma is not uncommon.

CLINICAL FINDINGS: SOUTH AFRICAN CASES

The cases in this country were identical except for the following:

1. *Diarrhoea and vomiting* were rare. Many of the patients gave a history of constipation.

2. *Peripheral Neuritis* was present in one case. Knee jerks were absent. The peripheral neuritis was thought to have no relation to the disease under discussion.

3. *Skin.* Exanthemata were not usually present. One child showed a rubeolar exanthem on the trunk and thighs. A naevus was seen in only one case.

4. *Glaucoma.* Eye symptoms were very common. Most of those affected complained of blurred vision, but the presence of glaucoma was not ascertained.

5. *Blood Pressure and Urine.* (*a*) *Urine.* The specific gravity and the amounts of urine passed were not measured in any of the cases. Albumin was not present in the urine of five patients examined. In a sixth patient a slight trace of albumin was detected, but the Acting District Surgeon who conducted these examinations stated that this patient was probably suffering from some other condition.

(*b*) *The Blood pressure* readings are interesting and are submitted here without comment for the writer has not seen such low diastolic pressures recorded and is not in a position at present to investigate medical literature to ascertain whether or not such low diastolic pressures have been recorded.

Patient	*Blood Pressure in mm. Hg*
1.	120/60
2.	130/20
3.	130/65
4.	120/20

These readings were taken three times.

A request that the specific gravity of the urine and further observations of the blood pressure of these patients be made, was not followed up, for by the time these requests reached the District Surgeon, all the cases had recovered.

POST MORTEM FINDINGS

There were four deaths in this outbreak. The deceased were: three coloured females, aged 31, 10 and 10 years; one coloured male aged 8 years.

Autopsies were performed on two of the deceased, namely the Coloured female aged 31 and the Coloured female aged 10 years.

In each instance the District Surgeon concluded that death was due to myocardial failure.

Only the main findings will be described.

Oedema. The adult Coloured female had slight oedema and the Coloured female child had very marked oedema of the legs.

Pericardial Sac and Heart. Straw-coloured fluid was found in the pericardial sacs of both of the deceased. In the child there were small haemorrhages on the pericardium and the external surface of the heart.

Large Blood Vessels. These were described as congested.

Pleural Sac and Lungs. In both patients these contained straw-coloured fluid varying in quantity from ¾-1 pint. The lungs were grossly congested but no consolidation could be detected.

Peritoneum and Peritoneal Sac. There was a small quantity of straw-coloured fluid in the peritoneal cavity of the Coloured female child.

Liver, Kidneys and Spleen. These were congested and in both cases there appeared to be some signs of liver degeneration.

The conclusion that death was due to myocardial failure was based upon the finding of transudate in both pleural cavities, congestion of lungs, and degeneration of liver.

Histological Findings. The second autopsy was conducted in the presence of the writer, who retained certain specimens for further investigation. These organs were unfortunately preserved in absolute alcohol as no 10% formalin was available at the time. They were delivered to the Government Chemical Laboratories in Johannesburg, but it was decided in view of the fact that there would be no prosecution under the Food, Drugs and Disinfectants Act or the Public Health Act, that they should be examined histologically by the South African Institute for Medical Research.

Dr. P. J. P. Bekker reported to the Secretary for Health on these specimens as follows:

'Specimens of liver, kidney, heart muscle and stomach were submitted for histological examination. In each organ post-mortem autolytic changes had proceeded to such an exten that precise histological detail had been obscured. This was particularly marked in the case of the stomach.

Section of the heart muscle shows the presence of segmentation and fragmentation. In addition the muscle cells show fatty change. The myocardial capillaries are widely patent and mostly empty.

Section of the liver tissue shows marked dilatation of the sinusoids and portal vessels. In addition there is focal fatty degeneration of the hepatic cells. The histological features are quite unlike those usually seen in the chronic venous congestion of congestive cardiac failure, but are compatible with an acute cardiac or peripheral failure.

Section of the kidney tissue shows the presence of slight dilatation of the glomerular capillaries and renal vessels generally, which are mostly empty. In addition there is tubular necrosis largely *post mortem* in origin. The interstitial tissues show no significant pathological change.

Section of the stomach shows autolysis of the mucosa. Nothing significant has been noted in the submucosa or muscularis. The subseral vessels show dilatation and are empty.

The above histological features taken in conjunction with the major autopsy observations of oedema of the legs, pleural and peritoneal transudates and congested liver and spleen indicate either acute myocardial or peripheral failure.'

The pathological findings in the Eastern cases may be summarised as follows:

1. Extensive vascular dilatation of the deeper layers of skin;
2. Atrophy of the myocardium;
3. Capillary dilatation of internal organs, especially the omentum;
4. The liver has a nutmeg appearance;
5. Vascular outgrowths resembling sarcoids are present;
6. Vascular changes are present in the ciliary body of the eye, leading to excess of fluids in the anterior chamber of the eye.
7. In addition the presence of oedema and fluid in the pericardial sac, pleura, peritoneum, as described in the post-mortem findings given above, were found.

It is felt that the following viscera should be submitted for pathological examination in the event of future deaths:

1. Heart and pericardial fat (portion);
2. Liver (portion);
3. Kidneys;
4. Portion of omentum;
5. Portion of skin and any skin growths;
6. Brain;
7. Eye.

Investigations in Loco. The owner of Farm A had already formed an opinion of the cause of death when the investigations were conducted on the 12 September 1947. Specimens of the wheat which had been consumed by the Coloured persons and by the poultry on Farm A were examined and found to contain seeds

which microscopically resembled those of the Mexican poppy.

A visit to a second farm (Farm C) on which there were Coloured persons also reported to be ill, revealed that these victims had oedema of the legs and complained of blurred vision.

The farmer denied that his Coloured labourers had eaten any wheat containing any of the seed. However, it was decided, despite the protests of the farmer, to examine the wheat which they were consuming. They had in their possession some ground wheat which was a portion of what they normally consumed. On compressing a sample in the palm of one hand with the fingers of the other, the presence of what appeared to be portions of the husks and the contents of the seed of the Mexican poppy were detected, and there is no doubt whatsoever that these patients were suffering from the same condition as those on Farm A.

The magistrate was later requested to instruct these farmers in writing to cease supplying to their Coloured labourers any wheat which contained any of the seed.

An inspection was made of the two local mills and it was found that these were fitted with efficient sieves and winnowing apparatus to extract the seeds of this plant. However, it was discovered that the farmers in the district were in the habit of collecting the rejects from these mills and using rejects for the purposes of feeding their Coloured folk and poultry. It also appeared that some of the farmers, not necessarily the two farmers whose farms were investigated, were in the habit of grinding their own wheat, or alternatively of giving wheat containing the seeds to the Coloured folk who ground, for their own consumption, the wheat together with the seeds.

The owner of one of these mills stated that he knew it to be a common practice for the owners of wheat to request the miller not to set the sieves too finely because they would lose too much wheat. This resulted in the wheat, which is rejected and collected subsequently, being heavily contaminated with the seeds of the Mexican poppy. The contaminated wheat was then used as is described above.

Further Investigations. Samples of the wheat were analysed. The College of Agriculture, Potchefstroom, found it to contain 13.5% of the seed of the Mexican poppy. The Chief of the Division of Botany and Plant Pathology, Pretoria, reported that the seed found in the wheat was microscopically almost certainly identical with the seed of the Mexican poppy. This opinion had been confirmed by Mr. Sellschop, Division of Agricultural Education and Research, College of Agriculture, Potchefstroom.

The question of culpability was also investigated but nothing could be done under the Food, Drugs and Disinfectant or the Public Health Acts, because the ground wheat containing the seeds of the Mexican poppy was not offered for sale to the public.

SUMMARY

1. This paper describes outbreaks of *Argemone Mexicana* seed poisoning on two farms in one of the North-Western Cape Districts.

2. The cases and death were confined to Coloured persons.

3. The paper compares the clinical findings and signs found in the cases which occurred in South Africa with those which have occurred in India and other Eastern countries.

4. The post-mortem findings are similarly compared.

5. Results of the inspections *in loco* prove that it is a common practice for farmers to feed to their Coloured labourers wheat contaminated with the seed of the plant.

RECOMMENDATIONS

It is felt that very active steps should be taken to inform all concerned of the danger of using wheat for human or animal consumption if such wheat is contaminated with the seeds of this plant.

The writer wishes to acknowledge his thanks to the Secretary for Health for permission to publish this paper and to Dr. B. M. Clark, Deputy Chief Health Officer, Union Health Department, and Prof. Douw Steyn, Department of Pharmacology, University of Pretoria, for their assistance in its preparation.

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POISONING WITH THE SEEDS OF *ARGEMONE MEXICANA* (MEXICAN POPPY) IN HUMAN BEINGS

INDIAN EPIDEMIC DROPSY IN SOUTH AFRICA

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Argemone Mexicana Linn., commonly called Mexican poppy, Texas poppy, prickly poppy, yellow poppy, thistle poppy, devil's fig, Bathurst weed, *steekbossie, bloudissel,* and (erroneously) Scotch thistle, is a native of tropical America and is of common occurrence in the Union, especially in the more arid regions. It is a typical weed (Fig. 1) being frequently common in and along water courses, on the edges of dams, and on cultivated lands. Phillips[2] describes it as a somewhat bushy annual herb growing up to 3 feet high; the stems which contain a yellowish juice, are prickly and each lobe of the much-lobed leaves ends in a spine; the

flowers, which are about 1½ inches in diameter, are white or yellow, and the fruit which is a spiny capsule, 1 to 1½ inches long and up to ¾ inch in diameter, contains

Fig. 1. *Argemone mexicana* Linn. in the fruiting stage.
Fig. 2. Seed capsule and seed of *Argemone mexicana.*

brown seeds, $\frac{1}{11}$ inch in diameter, globose, pointed at one end with a reticulated surface (Fig. 2).

Ghose[4] states that the suitability of the oil of *Argemone* seed for art is considered superior to that of poppy seed. The possibility of using *Argemone* oil in paints and varnishes has also been raised.[5]

In view of the cases of 'epidemic dropsy' which recently occurred in the Carnarvon district, it was deemed advisable to publish a detailed account of 'epidemic dropsy' as it occurs in India and also to publish the results of our experiments with the Mexican poppy in an attempt to prevent further cases of poisoning in human beings with the seed of this plant.

REVIEW OF THE LITERATURE ON 'EPIDEMIC DROPSY'

Epidemic dropsy has claimed many victims in India and even as early as 1926 suspicions about the cause of this disease fell on the so-called 'katakar oil' (oil of the seed of *Argemone Mexicana*). In the last 10 years most interesting and valuable researches into the possible causes of epidemic dropsy in India have been done by Chopra,[13, 15, 27] Lal,[8, 16, 20, 28, 30, 32, 33] Gupta and others. From their publications there appears to be no doubt that *Argemone* oil is the cause of epidemic dropsy in India. Contamination of stocks of mustard seed with *Argemone* seed takes place through accidental or wilful adulteration (Lal *et al.*[8]). The seeds of *Argemone Mexicana* bear a superficial resemblance to the black variety of mustard seeds.

Symptoms of Epidemic Dropsy in Man.[9, 11, 12, 13, 14] The incidence is highest in the third quarter of the year, most cases occurring in July and August (Ghose[9], Lal and Das Gupta[10]). Crops of mustard seed are reaped and gathered in March and are marketed in April. Considering that it takes one to two months for crushing the seed and expressing the oil and allowing approximately three days to a few weeks for the latent period of epidemic dropsy, it becomes clear why the incidence of epidemic dropsy reaches its peak in the third quarter of the year. Lal and Das Gupta[10] state that there is a secondary rise in incidence in spring for which there is as yet no satisfactory explanation.

The basic characteristic of epidemic dropsy is 'the dilated and engorged capillary angiomatous condition' (Tribedi and De[11]). The poison in *Argemone* seed appears to have a special predilection for blood vessels. There is an increase in capillary permeability and almost all the factors controlling the fluid exchange between the blood and the tissues are disturbed (Chopra *et al.*[13]).

Pasricha and his co-workers[12] state that in the differential diagnosis of epidemic dropsy and cardiac dropsy a point of importance is that in the former disease the sedimentation rate of the red blood corpuscles is increased while it is normal in cardiac dropsy. This phenomenon is in keeping with the observations made by Chopra *et al.*[13] and Tribedi and De,[11] viz. that the sera of epidemic dropsy patients have a relative viscosity and surface tension below those of normal subjects.

Pasricha *et al.*[14] state that 'the onset of aches and pains in the body and the development of oedema were considered as indicating toxic effects'. However, in cases of epidemic dropsy the symptoms commence with the appearance of sarcoids, mild diarrhoea, and slight oedema of the legs.

In epidemic dropsy oedema of the legs is the most common form, hence the name of the disease. There is flushing and hyperpigmentation of the skin, while red

spots and sarcoids, pin-point to ½ inch in size, may also be in evidence. These verrucoid eruptions may also appear on the mucous membranes. Diarrhoea is present in approximately 50% of the cases and the temperature may be elevated. Other symptoms are: palpitation, praecordial pain, dyspnoea, bleeding from the gums, sarcoids in the bowel, haemoptysis, epistaxis, and menorrhagia.

In chronic and recurrent cases the average blood cholesterol content is increased, while in acute cases it is also slightly elevated.[13] It is suggested that the increase in blood cholesterol 'may be a compensatory mechanism to increase the colloid-osmotic pressure of the blood which is markedly lowered in epidemic dropsy due to depletion of the albumin fraction'.[13] On the other hand, there is a fall in the serum calcium in cases of epidemic dropsy.

Toxicity of Argemone Oil for Man. In the various outbreaks, the Indian investigators found that the mustard oil used by the victims in their diet was contaminated with quantities of argemone oil varying from 1.0-5.7%. The victims had consumed on an average ½ to ¾ oz. of the contaminated mustard oil daily for 10 days or longer. Pasricha and his co-workers[14] state that the amount of argemone oil necessary to produce toxic effects in man is approximately 0.88 c.c. per 100 gm. of body-weight.

Post Mortem Appearances and Histology in Man. Apart from the lesions described above, the following are of importance:

Skin and Subcutaneous Tissues:[16] Typical are (1) a thick deposit of melanin in the deeper layer of the *rete*; (2) flattened-out papillae in some parts of the skin due to interpapillary oedema; (3) dilatation of interpapillary vessels associated with considerable perivascular infiltration (oedema); (4) bunches of dilated young capillaries all through the subcutaneous tissues and in the adipose layer; (5) in some parts of the subcutaneous tissues there is a large amount of extravasated blood.

INVESTIGATIONS INTO THE TOXICITY OF *ARGEMONE MEXICANA* AND POSSIBLE CAUSES OF EPIDEMIC DROPSY

(*a*) *Experiments on Animals.* As far as poisoning of animals by the Mexican poppy is concerned, Seddon and Carne[6] conducted experiments with the plant on sheep, while Hart[7] employed fowls. Furthermore, it is stated[18] that *Argemone* seed-cake, after expression of the oil, has deleterious effects upon animals.

Seddon and Carne[6] drenched sheep with (i) immature and mature *Argemone* seed pods; (ii) ground seed soaked in water overnight; (iii) an aqueous extract of 2 lb. of minced green leaves, and (iv) an aqueous extract of 2 lb. of minced green fruiting heads. In addition they fed sheep chopped-up leaves and minced fruiting heads of the plant in their daily ration, drenched a rabbit with 2.5 gm. of the mature seed, and drenched and injected subcutaneously guinea-pigs with aqueous extracts of the mature seed. In no case did any of the experimental animals develop any symptoms of poisoning. Unfortunately, the quantities of leaves, fruiting heads and seed fed, or drenched are so small that no reliable conclusions about the toxicity or harmlessness of the plant can be drawn from the negative results of the experiments conducted by Seddon and Carne.

In Australia Hart[7] investigated deaths in fowls which had consumed wheat containing seeds of *Argemone Mexicana*. He found that the experimental fowls ingested approximately 25.0 gm. of Mexican poppy seed in 9 to 12 days before symptoms of poisoning appeared. Two birds died after having eaten 60.0 gm. of the seed in 26 days and 62.0 gm. of the seed in 31 days, while other birds recovered after having consumed 168.0 gm. of the seed in 56 days. The animals exhibited the following symptoms: Oedema of the wattles, intermandibulary spaces and subcutaneous tissues, general weakness, diarrhoea and darkening of the wattles and comb. At autopsy the following were found: Oedema of the wattles and subcutaneous tissues, oedema of the lungs, hydropericardium, necrosis of the mucous membrane of the crop, ulcers on the lining of the gizzard, catarrhal enteritis, liver dark in colour.

As far as the cause, or causes, of Indian epidemic dropsy are concerned, three theories were originally advanced,[26] viz. (i) an infecting organism or virus, (ii) intoxication due to diseased rice, mustard oil, or an adulterant, and/or (iii) avitaminosis. It was rightly stated that diet probably played a rôle in the degree of susceptibility of victims (conditioned toxicity). Bhattacherjee[19] found a deficiency of vitamin D in the diet of those subject to epidemic dropsy and he states that there is a fall in the serum calcium with the appearance of dropsy. Pasricha[12] states that a diet high in rice appears to increase susceptibility to epidemic dropsy. From our knowledge of the role played by certain vitamins in the building up of a normal, healthy blood vessel system, and especially capillaries, it appears obvious that a diet deficient in *rutin* and vitamins C, P and E will render human beings and animals much more susceptible to epidemic dropsy, as the outstanding feature of this disease is a weakening of the capillary walls with consequent extensive oedema.

In an attempt to elucidate the causes of epidemic dropsy in India, Chopra *et al.*,[13, 15, 27] Pasricha *et al.*,[12, 14] Bhattacherjee,[19] and Lal *et al.*[20] conducted numerous experiments upon cats, guinea-pigs, rats, mice and human beings with argemone oil, pure mustard oil, and mustard oil contaminated with argemone oil. According to Pasricha and his co-workers[14] the amounts of argemone oil necessary to produce toxic effects in guinea-pigs, are: unheated oil 0.54 c.c. per 100 gm. body weight; oil heated to 170° C, 0.95 c.c. per 100 gm. body weight; and oil heated to 240° C, 1.23 c.c. per 100 gm. body weight.

Paricha *et al.*[12, 14] and Lal *et al.*[20] describe the following histological lesions in rats, guinea-pigs and mice fed with argemone oil: highly dilated capillaries engorged with blood in the dermis associated with perivascular infiltration (in rats); in one case a large number of new capillaries was seen in the heart, and most of the internal organs showed congestion; portal congestion and thrombosis of some of the tributaries of the portal vein; pronounced fatty degeneration of the hepatic cells

scattered throughout the lobules with small foci of necrosis near the peripheri; acute haemorrhagic glumerulo-tubular nephritis with pronounced degenerative changes in the convoluted tubules and descending limbs of Henle; in several areas the epithelium was detached in the form of tube casts; pronounced congestion of the intestines with extravasation and haemorrhages in some areas; oedema of the gastric and intestinal mucosa, the vessels of the villi being congested and the epithelial cells swollen and desquamated.

In rats on pure mustard oil there was no vascularization and no sign of oedema but only engorgement of the vessels of the heart, lung and kidneys.[20]

(*b*) *Experiments upon Human Beings.* Chopra[13, 27] and Lal and their co-workers[28] fed human beings with food prepared with mustard oil which gave a positive test for argemone oil. These volunteers developed symptoms of epidemic dropsy within two to four weeks. Controls whose food was prepared with mustard oil which gave a negative test for argemone oil, developed no ill effects.

From their observations in the field and from the results of chemical tests and experiments conducted by them on human beings and animals, the above investigators concluded that mustard oil, accidentally or wilfully, adulterated with argemone oil is the cause of epidemic dropsy in India. As a result of their findings Lal *et al.*[30] recommended that the sale of mustard oil containing 0.5% or more of argemone oil for human consumption should be prohibited. However, Sarkar [22, 23, 24, 25, 26, 29] was opposed to this view held by all other investigators. He[22] stated that the amount of argemone oil necessary to induce epidemic dropsy is not present in potent mustard oil samples from affected areas. He maintained that the cause of epidemic dropsy must be some other contaminant of mustard oil. He[23] furthermore stated that 'mustard oil prepared from argemone-free mustard seed undergoing microbial decomposition and resembling samples of oil reputed to have caused symptoms of epidemic dropsy in man, is fairly toxic to rats'. Sarkar[24] moistened argemone-free mustard seed and inoculated it with the fungus *Monilia* which infects 'Ghani' cakes (prepared from mustard seed) when kept in damp places. When this infected mustard seed showed good growth of the fungus, the oil was expressed from it. This expressed mustard oil gave a positive reaction with Lal's nitric acid test. As a result of this experiment, Sarkar contended that if this test were a criterion of the toxicity of mustard oil, these fungus-infected mustard seeds without the admixture of argemone seed, or argemone oil, may be the causal agent of epidemic dropsy. In spite of Sarkar's difficulties in accepting argemone oil as the cause of epidemic dropsy the results of the extensive field and laboratory investigations conducted by Chopra and Lal and their co-workers in India and the results of Hart's and our own experiments upon fowls, the overwhelming evidence at our disposal certainly points to argemone seed (or oil) as the cause of epidemic dropsy.

(*c*) *The Poisonous Principle of Argemone Seed or Oil.* In the earliest investigations[31] it is stated that *Argemone Mexicana* contains the alkaloids *protopine* (*argemonine*) and *berberine*. Henry[31] states that *berberine* is moderately toxic to the larger animals. In the rabbit it causes dyspnoea, cardiac damage, lowered blood-pressure and paresis. Haemorrhages and congestion of the lungs are found *post mortem*; renal damage has also been reported. *Berberine* has a bitter taste and it is used in the treatment of oriental sore. *Protopine* has a local anaesthetic action and Henry[31] states that, in small doses 'it has a narcotic action in frogs, while large doses abolish reflex activity and show a *curare-like* action. At times there is evidence of medullary stimulation. In mammals *protopine* causes epileptiform, camphor-like convulsions; but unlike camphor, these are associated with damage to the circulation'.

In 1939 Lal and his co-workers[6] reported that 'a white crystalline substance (w.c.s.) had been isolated from the oil of *Argemone Mexicana* and also from a proved toxic oil but not from pure mustard oil', and that the role of this w.c.s. in the causation of epidemic dropsy had as yet not been determined. In 1941 Lal and his co-workers[32] referred to the presence of a crystalline free base (c.f.b.) in addition to the w.c.s. which were both isolated by Mukherji and his co-workers. Both these chemical substances were biologically active, inducing in rats the essential biological changes of epidemic dropsy in man. Exposure of argemone oil to light for a sufficiently long time, or the removal of the c.f.b. from argemone oil, renders the oil biologically inactive.

Owing to the destructive effect of light on the poisonous principle contained in argemone oil, it is recommended that samples of mustard oil suspected of containing argemone oil, be submitted in coloured phials. In a further publication these authors[33] state that (1) 'the fraction obtained from argemone oil by the process of saponification and extraction with hydrochloric acid gas has not proved toxic to man although it reacts specifically to chemical tests and induces essential histological changes in white rats; and (2) the residue of argemone oil left after extraction of the fraction with hydrochloric acid gas is non-toxic to man'. They concluded that the methods of extraction so far employed, isolate only a portion of the toxic radicle and that neither of the split products is toxic.

Mukherji and Mathur[34] state that the empirical formula of the toxin (the white crystalline substances) is $C_{19}H_{15}O_4N$. They described some chemical reactions (colour tests) of this toxin (w.c.s.) as compared with *berberine* and *protopine* and showed that the w.c.s. is not identical with either *berberine or protopine.*

Heating argemone oil up to 150° C had no effect on its degree of toxicity, whereas heating the oil to 250° C. for 15 minutes almost completely destroyed its toxicity.[12, 14]

Sarkar[25] stated that Lal's nitric acid test is not specific for argemone oil as in his (Sarkar's) tests it also gave positive results with nine other vegetable oils (e.g. linseed oil, olive oil, jute seed oil, sesame oil, etc.), all of which are common adulterants of the mustard oil of commerce. Sarkar added that these nine oils gave negative results with his ferric chloride test. According to Sarkar, argemone oil heated with

ferric chloride solution in the presence of concentrated hydrochloric acid and alcohol gives an orange-red precipitate. This test detects argemone oil in 0.75% concentration. He added that 'three reputed dropsy-positive samples of mustard oil failed to give positive argemone tests'.

Sarkar and Rahman[35] extracted from argemone oil alkaloids which are not very toxic when administered orally to young rats in daily doses of 1.0 mg. of their hydrochloride. These alkaloids extracted with ferric chloride, or cold dilute hydrochloric acid, produced 50% mortality in 30 and 44 days respectively, and the alkaloids obtained by precipitation as picrates caused 50% mortality in 22 days with a daily dose of 0.5 mg. They concluded that these results, however, do not necessarily prove that argemone oil is the cause of epidemic dropsy.

EXPERIMENTS WITH THE ARGEMONE PLANT AND ITS SEED AT ONDERSTEPOORT AND AT THE UNIVERSITY OF PRETORIA

In 1936 one of us (D. G. S.)[3] fed the fresh plant in the pre-flowering and early flowering stage to sheep at Onderstepoort. They consumed up to 11 lb. in five days without developing any symptoms of poisoning. Burtt-Davy,[1] however, stated that 'this plant has an acrid, yellow juice, and is poisonous'. The above negative result obtained at Onderstepoort is no proof that the plant is non-toxic as even the most toxic plants vary considerably in their degree of toxicity.

After the outbreak of epidemic dropsy in the Carnarvon district in 1947 a quantity of mature Argemone seed was obtained from the lands on which the 'poisonous wheat' was grown. As Hart[7] had produced symptoms of poisoning in fowls, closely resembling those of epidemic dropsy in man, by feeding them with Argemone seed, it was decided to use these birds in the investigation into the possible toxicity of the Argemone seed which was suspected of having caused the Carnarvon outbreak of epidemic dropsy. The mature seed was fed to six White Leghorn cocks. The seed was ground and fed daily mixed in the balanced ration (mash) of the birds. Prof. A. M. Gericke, of the Department of Poultry Husbandry, University of Pretoria, kindly placed the birds at my disposal and rendered assistance in the execution of the feeding experiments.

Symptoms of Poisoning. The most conspicuous symptoms of poisoning exhibited by the birds were: pronounced yellowish-green diarrhoea, apathy, general weakness which progressed until the fowls were unable to stand, pronounced oedema of the comb and wattles which eventuaully showed localized areas of dry necrosis, oedema of the pericardial, intermandibulary and subcutaneous tissues, especially marked over the sternum, and loss in weight. The combs became dark-purple in colour and the affected birds, when walking, lifted the legs high without bending the leg-joint.

Post Mortem Appearances. Hyperaemia of the lungs; heart markedly dilated and flabby; pronounced oedema of the intermandibulary and subcutaneous tissues (over the sternum), and of the comb and wattles; dry necrosis of the comb commencing at the tips; localized necrotic areas on the oedematous wattles; oedema of the pericardial tissues; subacute or chronic catarrhal gastro-enteritis; and hyperaemia of the liver which also showed degenerative changes associated with varying degrees of cirrhosis.

The above symptoms and post mortem appearances are very similar to those described by Hart[7] in his experiments with Argemone seed on fowls, and they also bear a close resemblance to the disease known as 'Indian epidemic dropsy' in man, alleged to be caused by mustard oil contaminated with argemone oil.

Histology. Specimens of liver, kidney, spleen and myocardium of the fowls used in the above experiments were kindly examined by Dr. H. P. de Boom of the Department of Pathology, Onderstepoort Laboratories, and the following report was received from him:

'Liver, kidney, spleen and myocardium were microscopically examined. In the case of the liver a subacute hepatitis was diagnosed. Slight, peripherally situated vacuolar changes were observed in the parenchyma, not the extensive hydropic degeneration as described by Pasricha, Lal and Banerjee (1940) with fairly pronounced perilobular accumulations of lymphocytes and monocytes, as well as pseudo-eosinophiles. Early collagenisation of the intralobular reticulum was noted. The bile duct epithelium revealed fatty changes only in those cases receiving the larger quantities of seed. The most remarkable lesion was the accumulation of large lipid-containing histiocytes in the adventitia of the branches of the portal vein, and in the connective tissue of the portal tracts. (Staining affinities of this substance contained in the histiocytes: light brownish red with Sudan IV, gray with Giemsa, not fuchsinophile.)

In the kidneys a slight focal interstitial nephritis was found. The spleen was normal. The myocardial scar observed in one case probably had no connection with the administration of the seed; nevertheless this lesion should be borne in mind with reference to the clinical picture in the human.'

TREATMENT OF CASES OF EPIDEMIC DROPSY

As (*a*) one of the most characteristic lesions in epidemic dropsy is damage to the capillary walls, and (*b*) the poison in Argemone seed (or oil) appears to have a special predilection for blood vessels, it is obvious that the chief objective in the treatment of this disease should be to protect and restore the damaged capillaries. This can best be done by the administration of liberal quantities of *rutin* and vitamins C, E and P. In addition, the patient should be placed on a diet rich in minerals and vitamins, especially the B complex (brewer's or food yeast), C. D. E and P. Furthermore, it is of the utmost importance to protect and stimulate the detoxifying capacity of the liver. This should be done by placing the patient on a diet rich in protein (approximately 20%, mostly of plant origin) and starch (approximately 80%), and low in fat (approximately 6%). The patient should also be given the following:

(*a*) Five ounces of glucose *per os* and 10 units of insulin twice daily.

(*b*) Vitamins A, B complex (brewer's or food yeast),

C and D. It is advisable also to supplement vitamin K as there is a likelihood of deficiency in this vitamin when liver function is impaired. The administration of liver extracts is also recommended.

(*c*) In all cases of poisoning (except in poisoning with digitalis-like substances) intravenous or intramuscular injections of a 10% solution of calcium glucono-galacto-gluconate (Calcium-Sandoz) are advisable. (A 20% solution of this calcuim preparation should not be injected intramuscularly as it is too irritant.) As calcium deficiency has been reported in cases of epidemic dropsy, the administration of preparations containing this substance will probably be of great benefit to the patient.

(*d*) Two to four grammes of methionine daily in the food. As choline plays a rôle in preventing fatty degeneration of the liver, its use is recommended in cases of damage to this organ. Choline and methionine are very expensive and casein, which contains both these substances, may be used instead.

(*e*) *Glutathione*. This substance is almost universally present in animal tissues but is concentrated in the liver. It contains an active sulphydryl (–SH) group and acts as a respiratory carrier of oxygen, and consequently plays an important rôle in the detoxifying processes in the liver. For this reason its administration is recommended.

Apart from the above treatment, which aims at protection and strengthening of the capillariés and the liver, any symptomatic treatment which may be indicated, should be applied. In view of the heart affection, cardiotonics are indicated.

Manson-Bahr suggests that egg-and-milk albumin are indicated in large quantities, and that treatment should then be directed to giving a Thyroid Ext. and Tr. ephedrae 20-30 mins. with calcium lactate 10 grms. three times daily.

DISCUSSION AND RECOMMENDATIONS

Suspected cases of epidemic dropsy have been investigated by Dr. R. E. Meaker in the North-western Cape districts. In all its aspects the disease resembled the so-called Indian epidemic dropsy. In experiments conducted upon fowls with the seed of the Mexican poppy collected from the lands on which the wheat eaten by the victims was grown, symptoms and histological lesions similar to those seen in human epidemic dropsy were produced. With the information at our disposal we can come to no conclusion other than that the said outbreak in the Carnarvon district was epidemic dropsy caused by the consumption of wheat contaminated with the seed of the Mexican poppy.

This matter was immediately brought to the notice of the Department of Agriculture and the Department of Health and it was recommended that either *Argemone Mexicana* be proclaimed a weed under proclamation No. 117 of 1941 in pursuance of the Noxious Weed Act, No. 42 of 1937, as amended, or failing this, to distribute pamphlets warning wheat-growers about the danger of the presence of the seed of the Mexican poppy in wheat or, for that matter, in any food product grown on lands infested with this poisonous weed. However, the Secretary for Agriculture did not consider it necessary to proclaim the plant a noxious weed and stated that to ensure the removal of Argemone seed from wheat 'every wheat mill in the Union has been, or is being equipped, with the necessary apparatus to remove all such seeds from the wheat before it is milled'. Furthermore, the Secretary for Agriculture suggested that the plant be eradicated by the new weed-killer 2, 4-D.

The writer knows only too well, from personal experience, that the wheat mills in our country are equipped with the necessary winnowing appliances for removing the seeds of weeds which may be present in wheat. However, in the course of investigations into outbreaks of bread-poisoning, he visited the mills where the wheat contaminated with the seeds of poisonous weeds had been ground. In some cases he found that the wheat was heavily contaminated with the seeds of all kinds of weeds and that a high percentage of these seeds was ground with the wheat. The writer discussed this matter with the owners of the mills concerned and they informed him that the owners of the wheat had requested them not to set the sieves "too finely" because they would lose too much wheat. It is precisely in these families which made these requests to the millers that cases of bread-poisoning occurred. Furthermore, the writer warned many families of the danger of eating bread prepared from meal contaminated with the seeds, or other parts, of weeds (e.g. Senecio, Argemone, etc.). On some farms, and at some mills, in the Eastern Cape Province he saw bags of wheat, which were ground for domestic use, so heavily contaminated with weed seeds that they contained only about 20%-30% of wheat grains. In every case where he warned the families concerned, their reply was: 'We are poor and cannot afford to lose some of our wheat as we have to sell the "clean wheat" and use the contaminated wheat ourselves.'

Another point of great importance is that many of the lesser-privileged European and non-European wheat-growers do not send their wheat (which is contaminated with the seeds and other parts of weeds and which they retain for their personal use) to be ground at mills, but grind it themselves, or they cook the contaminated wheat and use it instead of rice.

From the above it is clear that *with the best and most efficient winnowing appliances in the world, we shall not be able to protect the impecunious wheat-grower (European and non-European alike), and it is precisely these unfortunate people who fall victims to bread-poisoning (senecio-, argemone-poisoning, etc.). The efficient winnowing appliances protect only those who can afford to use, or buy, wheat free from weed seeds.*

The writer feels that it is our duty to warn and protect the ignorant and poor European and non-European wheat-grower, and, above all, to protect their innocent children. As far as we see this problem, it is not necessary for these lesser-privileged people to spend money on modern weed-killers. As a rule their wheat lands are of such a limited size that they and their families can easily manage to eradicate *Argemone Mexicana* by hand (hoeing, etc.). All we need do is

to warn wheat-growers of the danger of the presence of the seed of this weed in wheat, or in any other crop, and to enforce its eradication.

SUMMARY

A review of the history, symptoms, post mortem appearances, and histology of epidemic dropsy in India is given. Investigations made by Indian workers in order to ascertain the cause, or causes, of epidemic dropsy have also been discussed. From the results of their investigations it appears reasonable to accept that mustard oil, wilfully or accidentally, adulterated with argemone oil is the cause of epidemic dropsy.

Our investigations into a fatal disease which occurred in the Carnarvon district and which resembled Indian epidemic dropsy in all its aspects, fully support the conclusions of the Indian investigators, namely, that epidemic dropsy is caused by the eating of food contaminated with the seed, or oil, of *Argemone Mexicana*.

Methods of treatment and prevention of epidemic dropsy are described.

As this dangerous plant is such a very common weed on cultivated lands on which crops for human and animal consumption are grown, it is urged that immediate steps be taken to enforce its eradication.

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ENCEPHALITIS AFRICANA

A PRELIMINARY REPORT*

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The terminology used is deliberately provocative. As the disease to be discussed has been named *Encephalitis africana*, it is not maintained at this stage that it is caused by one particular virus or a virus as yet unknown. The term is used as a starting point from which to commence further laboratory investigations. It is necessary, however, that the disease be recognized as early as possible by all practitioners and more especially by the general practitioner, as then, and only then, will it be possible to isolate the virus from the man. At the same time a plea is put forward for research work to be started on the isolation of viruses from mosquitoes and ticks collected from different areas in the Union. If these experiments prove successful, then neutralizing tests might indicate which viruses attack the population of the Union, causing the widespread occurrence of encephalitis. If this address could convince the profession of the widespread existence of this disease within the Union of South

* Presidential valedictory address to the Southern Transvaal Branch (M.A.S.A.) on 17 January 1950.

Africa, then early recognition will become easier and facilitate the investigations still to be performed in years to come.

In giving a clinical description of the disease, it is hoped that the goal set may be attained eventually, not only in the recognition of the cause, but also the treatment to be instituted. The clinical research presented is one collated over a period of 19 years. The conclusions reached are drawn from the material of about 100 cases, all of which have been seen in the acute stage.

Before discussing the disease as it manifests itself, it would be wise to enumerate those viruses which have been found to exist on the continent of Africa. Research work was commenced by the Rockefeller Institute in Central Africa at Entebbe in Uganda. The first publication from there came in 1940 from Smithburn, Hughes, Burke and Paul and Smithburn and Jacobs, who reported the isolation of the *West Nile virus*. This virus was isolated from an African woman in the area. In 1944 Smithburn and Haddow announced the isolation of the *Semliki Forest virus* from mosquitoes collected in the Bundinyama area in Western Uganda. This was followed in 1946 by the isolation of the *Bunvamwera Forest virus* by Smithburn, Haddow and Mahaffy. The virus was isolated from mosquitoes in the Bwamba area.

Lastly in 1948 Dick, Best, Haddow and Smithburn isolated a virus originally from mosquitoes, which they named *Mengo virus*. The discovery of this virus was important because the first author, Dick, developed an encephalitis during the first two days of which this particular virus was then isolated from his blood. The encephalitis was preceded by ' bronchitis '. On recovery from this bronchitis syndrome the encephalitic syndrome became manifest. The incubation period was estimated at 5 to 9 days and the duration of the disease was about 4 weeks. During the acute stage of the disease temperature rose to 101.8° F.

Surveys with neutralization tests indicated further that St. Louis virus and Japanese B virus must have been extensively active: the *St. Louis virus* in Kenya and the Congo and possibly the Sudan, and the *Japanese B virus* in the Sudan and the Congo. By the same method the *West Nile virus* was found to have been active in the Sudan, Uganda, Kenya and the Congo. This, to date, is all that is known of the viruses in Africa causing or liable to cause an encephalomyelitis.

Coming now to the clinical findings of the African encephalitis, it should be noted that it is not possible to give a description of a typical case as the disease appears in numerous clinical forms with all kinds of borderline variations between the different groups. It will be easiest to divide the clinical manifestations into seven large groups, bearing in mind the fact that borderline variations and mixed variations occur. The main seven groups are then:

1. Cephalalgic type.
2. Ocular type.
3. General neurological type.
4. Apoplectic type.
5. Cerebellar type.
6. Psychotic type.
7. Pseudo-tumour type.

1. *Cephalalgic Type.* In cases afflicted with this type, the only complaint is one of headaches. This headache may be periodic and severe or continuous and rather mild but with severe exacerbations, or continuously severe. The duration may be over a short period of weeks or over prolonged periods of many months. Severe attacks may be associated with biliousness and/or vomiting. There is, however, one common factor in all these cases, viz., that the patients are always able to indicate a particular month in which the headaches commenced. This factor is the salient point in coming to a tentative conclusion as to the presence of an encephalitis. Furthermore, variations in pupillary size and reaction may be present. Neurological manifestations otherwise are always absent. In this instance the diagnosis can never be confirmed without a lumbar puncture. The pressure of cerebrospinal fluid is very variable from low to approximately 550 mm. of water, the average being between 200-250 mm. of water. The cell count in the fluid varies from 15-500 cells per c.mm. and is lymphocytic in type. The diagnosis of encephalitis in this type is usually missed due to the fact that there are no further complaints or signs which make one aware of the seriousness of the condition, or which would urge one to perform a lumbar puncture.

2. *Ocular Type.* In the ocular type the patient complains of diplopia. On examination one finds pareses of the ocular muscles caused by lesions of the oculo-motor or abducent nerves, or combined lesions of both, with pupillary changes in size and reaction. Onset of signs is rapid, but variable in intensity. Again the diagnosis is difficult and cannot be verified unless lumbar puncture is performed for manometric pressure tests and cerebrospinal fluid examination.

3. *General Neurological Type.* In this type, as the name implies, general neurological symptoms are present in that pareses of extremities develop associated with reflex anomalies, paresis of one or several cerebral nerves may be present, disturbances of the different qualities in the sensory sphere may be found. From the psychiatric point of view there is usually mild irritability and mild impairment of the tenacity and/or retention. All the above may be associated with headaches and on occasion with neck rigidity in the early stages. The salient feature is, however, the variability of the signs found from day to day and even from hour to hour. The onset of the symptoms may be either gradual or sudden.

4. *Apoplectic Type.* In this type the onset may be over a period of 3 to 4 days or per-acute as with apoplexia. In other words, the condition resembles that of a thrombosis cerebri or an apoplexia. If the condition develops in a young individual with no cardio-vascular disease the diagnosis tends to be one of encephalitis. Lumbar puncture in these cases will mostly resolve any doubts. In older and aged individuals with or without cardio-vascular disease a lumbar puncture should be performed before coming to a conclusion. If one finds an increased intracranial pressure

and a lymphocytic pleiocytosis then the diagnosis of encephalitis is probably the correct one. The course of the disease in these cases is prolonged. It should be noted that variations in intensity of signs in this instance are not present.

5. *Cerebellar Type.* This type is rare, and if found is usually present in children. The neurological signs indicate an affection of the cerebellum to a gross degree. Signs may be variable in intensity and an admixture of cerebral symptoms may be present. Headaches of varying intensity are usually present.

6. *Psychotic Type.* In cases of this type the clinical picture is one of purely mental manifestations. The condition may resemble one of the major psychoses, i.e. schizophrenia in its different forms, manic depressive psychosis in its different phases or that of a mixed psychosis. It may also resemble a confusional state. The detection of any reflex variations or anomalies should make one very suspicious and averse from accepting the diagnosis of a psychosis. In cases with a clear family and personal history, and rather sudden onset, the diagnosis of a pure psychosis should be doubted and the diagnosis of encephalitis should be borne in mind. Again, as in the other types the performance of a lumbar puncture with manometric pressure tests and cerebrospinal fluid examination is the only method of arriving at a correct conclusion.

The type of mental manifestation is probably determined by the pathoplastic factor, i.e. pre-existing character factors. The pathogenic factor then causes the pathoplastic factor to become manifest. In general these patients recover completely after a period of 3-6 months.

7. *Pseudo-Tumour Type.* In this type, apart from general neurological symptoms either localized and focal, or variable and widespread, the salient features are those of increased intracranial tension especially the appearance of choking of the discs. In some cases one actually cannot come to a definite conclusion without the aid of a ventriculography.

OCCURRENCE OF SYMPTOMS IN PERCENTAGES

As it is not possible to describe one particular entity it is interesting to note the symptoms and express them in percentages of occurrence in the cases observed.

Initial gastro-intestinal, 3.3%.
Initial respiratory tract, 19.4%.
Temperature, 13.2%.
Headaches, 66.7%.
Diplopia, 28.9%.
Vomiting, 19.1%.
Sudden unconsciousness, 11.3%.
Epileptic seizures, 12.6%.
Neurological findings, 58.7%.
Hemiplegia, 1.8%.
Hiccough, 1%.
Positive Kernig sign, 9.2%.
Cerebellar signs, 6.4%.
Mental disorder, 21.6%.

Cerebrospinal Fluid. a. Pressure: The pressure of the cerebrospinal fluid is usually increased, the readings averaging 200 mm. of water. In some cases however there appears to be a low pressure state with the readings down to an average of 50-60 mm. of water. The highest level found was about 500 mm. water.

b. Cerebrospinal Fluid. i. The cell count in the cerebrospinal fluid varies from the normal 3-4 cells to 500 cells per c.mm. The average is however from 15 to 50 per c.mm. The pleiocytosis is of a lymphocytic type. On rare occasions a few polymorphonuclear cells are seen, but they are never in preponderance. The cell count returns to the normal within a period of 4 weeks to 3 months. On rare occasions the cell count remains increased for a period of years. One case observed and checked over a period of six years still exhibits a lymphocytic pleiocytosis.

ii. *Total Protein.* The total protein content in the early stages remains normal. There appears to be a tendency for the total protein content to increase above the normal values when the pleiocytosis in the cerebrospinal fluid disappears. After some weeks this total protein content then gradually returns to the normal level.

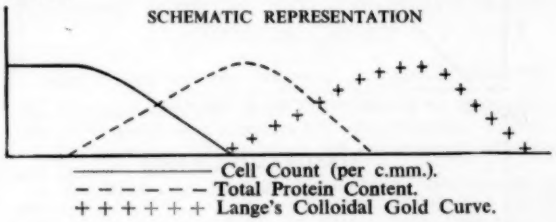
iii. *Colloidal Gold Reaction.* The Lange's colloidal gold curve remains negative until the total protein content commences to return to the normal level. At this stage a middle meningeal curve may develop. At the end of the disease therefore the only positive finding may be a middle meningeal curve with a normal cell count and a normal total protein content.

iv. The chloride content (as NaCl) remains normal throughout.

v. The glucose content in general remains normal but on rare occasions a decrease has been noted.

vi. The Wassermann reaction without any exceptions was negative.

In general, therefore, if one takes into account the cell count, total protein content, and colloidal gold curve (Lange) one could draw the following curves as a schematic representation of what has been indicated above:



One should remember, however, that there are several variations. One encounters cases where the pressure is increased but the cerebrospinal fluid is normal in all respects. Then one finds cell count and the total protein content increased, but the colloidal gold curve negative. Also further variations occur.

Temperature. Increase in temperature above the normal is rare. Usually the disease runs its course without any variations above the normal.

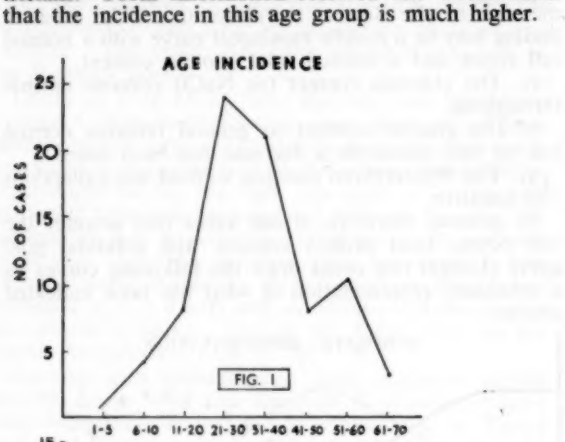
Prodromal Period. In many cases the outbreak of the encephalitic illness is preceded by an acute pyrexial condition usually diagnosed as influenza. This period precedes the encephalitic manifestation by about 14 days. This period is characterized either by the signs of an enteritis with diarrhoea (usually in children) or by signs of an affection of the respiratory tract in the nature of a tracheitis or bronchitis (usually in adults).

It is interesting to note the sequence of events in the individual suffering from the meningo-encephalomyelitis. However, once the temperature settles down before the outbreak of the encephalitic syndrome, it is rare for the temperature to recur, even in the initial stages of the encephalitis.

This is where the *African encephalitis* differs from most of the other forms of encephalitis known, as in all instances in the acute stage high temperatures are described.

Blood Count. In all instances where blood counts were performed no variations from the normal were ever found.

Age Incidence. The bulk of the people attacked are between the ages of 20 to 40 years, with a minor increase in incidence in the age group of 60 years. It would appear that the incidence in the age group of 1 to 20 is low. This may be a fallacious supposition as it should be noted that the writer has not seen many children in the age group of one to fifteen years, as these were distributed amongst the practising paediatricians. From information received one would think that the incidence in this age group is much higher.



Seasonal Incidence. The work performed indicates that odd cases keep cropping up throughout the year. It is evident, however, that the majority of cases occur in the months of November and December (early summer) with a sharp decline in January.

In coming to the above decision it should be noted that the date of onset of the condition was not the date of examination of the patient but the date of onset of symptoms.

Death Rate. The death rate is approximately 15% for all age groups combined but in the age groups of 50-70 years the mortality rises sharply for those age groups to just over 30%.

Course of the Disease. In the great majority of cases the disease seems to be self-limiting. The patient usually recovers within a period varying from four weeks to three months. There are, however, instances where the patient to all intents and purposes has fully recovered, and has no complaints at all, but the cerebrospinal fluid remained positive for a period of six years.

Sequelae. The majority of cases recover completely. Others complain of mild headaches and irritability as a permanent after-effect. In some cases concentration and memory seem to be mildly affected. A fairly frequent, but late manifestation is the development of *parkinsonism.* In only a few cases has this syndrome been seen to develop immediately after the acute stage of encephalitis. It has been noted that both in private practice and at the Out-Patient Department for Neurology and Psychiatry of the Johannesburg General Hospital the numbers of parkinsonistic syndromes in the young is extremely high.

Furthermore, epilepsy in its different forms is a well-established sequel of African encephalitis; 7% of the cases seen developed epilepsy from immediately to three years after the acute stage of encephalitis. Epilepsy need not necessarily be a sequel to encephalitis even if seizures do occur during the acute stage. On the other hand, if in the acute stage no epileptiform seizures occur, the patient may still develop epilepsy at a later stage. The prognosis should therefore always be very guarded. In rare cases the development of criminal and asocial tendencies has been seen. In children the development of a profound mental deficiency, in some cases associated with epilepsy has been observed. Lastly, four cases with the combined sequel of Parkinsonism and Epilepsy have been observed.

The following reasons indicate that African encephalitis may be caused by a virus different from those already described.

1. The known types all exhibit a high temperature in the acute stage. The African encephalitis rarely exhibits a temperature.

2. The mortality rate in other types is high varying from 30% to 70%. The mortality rate in African encephalitis is about 15%.

3. The sequel of parkinsonism is rare in all types described except for the virus 'A', i.e. encephalitis lethargica. In African encephalitis the incidence of the parkinsonistic syndrome as a sequel is high.

4. The sequel of epilepsy is not described in any of the other types of encephalitis. One case of Jacksonian epilepsy after St. Louis encephalitis is mentioned in the literature. Furthermore, Cecil in his handbook states that epilepsy may be a sequel to encephalitis but does not specify the type. Erasmus in his recent publication on epilepsy (*S. Af. Med. J.*, 29 October 1949) also indicates the possibility of epilepsy after African encephalitis.

Prognosis. Notwithstanding the fact that African encephalitis seems to run a milder course associated with a low mortality rate, the prognosis should always be guarded, especially with a view to the sequelae which may develop.

Treatment. There is as yet no known therapy. Various methods of treatment including the sulphonamides, penicillin, streptomycin, etc. have been used without success.

Recently three cases were treated with aureomycin. In the first case the cerebrospinal fluid was positive for a period of three months, including one specimen taken immediately prior to the commencement of treatment. The subjective complaints disappeared within 14 days of cessation of treatment and the cerebrospinal fluid returned to the normal in all respects. This patient who had been absent from work during the whole of this period returned to his duties immediately and has remained well to date—3 months later. The second case was one of encephalo-myelitis with a positive cerebrospinal fluid over a period of 6 months. After treatment the patient had improved subjectively though the objective neurological picture had not changed. The cell count in the cerebrospinal fluid had returned to the normal, the total protein was normal, but the colloidal gold test gave a meningitic curve. The third case did not respond in any way at all. The result attained in the first two cases indicates the possibility of future effective treatment.

Method of Transmission. Nothing is known of the method of transmission of the African encephalitis. Bearing in mind that in other forms of encephalitis, ticks and mosquitoes are known to be the transmitters of the disease, it is perhaps not unreasonable to suspect a similar method of transmission in the case of African encephalitis.

Immunity. With one exception, no cases were observed either with a relapse or re-infection after recovery. In the one instance the patient was seen in 1938 in an acute stage. He recovered and the cerebrospinal fluid had returned to the normal. The patient returned to work, and later enlisted in the army. He was then seen again in 1943, once more in an acute stage. The cerebrospinal fluid was again positive. The patient recovered completely and returned to duty. It is not possible to say whether this was a relapse, or a re-infection. If it was a re-infection it may well have been with another virus. All in all it would appear however that an immunity does develop.

Field-Marshal the Right Hon. J. C. Smuts, addressing the British Association for the Advancement of Science on *Science from the South African Point of View* said: 'The value of a hypothesis often depends not so much on its correctness as on its fruitfulness. A hypothesis may in the light of further knowledge prove to have been wrong, but in the meantime it may perhaps have suggested new points of view and new methods of attack and thus have cleared the way for real advances.'

May this apply to the hypothesis expounded above.

PASSING EVENTS

Mr. James Cuthbert, Specialist in Plastic Surgery, has moved his consulting rooms and is now in practice at 143, Lister Buildings, Jeppe Street, Johannesburg. Telephone 22-4100.

* * * *

Dr. D. Harbert Anthony and Dr. Daniel F. Fisher of 1718, Exchange Building, Memphis, Tennessee, United States of America, would like to receive from those of our readers able to assist them, reprints of or references to any paper they have written dealing with fracture-dislocation of facial bones, particularly orbital rim or floor and middle third of face, causing traumatic enophthalmos, diplopia, or sinus infection.

Mr. Arthur J. Helfet, F.R.C.S., has changed his address from National Mutual Buildings, Church Square, Cape Town, to 808, Grand Parade Centre, Castle Street, Cape Town. Telephone: Rooms: 3-2409; Residence: 6-8527.

50TH CHEMISTS' EXHIBITION, LONDON, ENGLAND

The Exhibition will be held this year in the New Hall of the Royal Horticultural Society, Westminster, S.W.1., from 3-6 October. The management (The British & Colonial Druggist Ltd.) will be pleased to welcome members from overseas who will be admitted on presentation of their cards. The Exhibition will reflect the latest advances in ethical preparations, pharmaceuticals, perfumery, toilet preparations, etc.

AMERICAN SOCIETY FOR THE STUDY OF STERILITY

The Sixth Annual Conference of the American Society for the Study of Sterility will be held on 24 and 25 June 1950, at the Sir Francis Drake Hotel, San Francisco, California.

The Secretary-Treasurer of the Society is Dr. Walter W. Williams, Springfield, Mass., U.S.A.

Copies of the 1946 Transactions of the Society were published by the Western Journal of Surgery, Portland, Oregon, and may be obtained through them. Price $6.00.

The 1947 Transactions were published by the Society, and may be obtained through the Secretary's office, 20 Magnolia Terrace, Springfield, Mass. Cloth bound, $6.00.

The Transactions for 1948 and 1949 will be found in early issues of *Fertility and Sterility*, the new Journal sponsored by the Society. Subscriptions may be secured from Paul B. Hoeber, Inc. (Medical Book Dept. of Harper and Brothers), 49 East 33rd Street, New York, N.Y. Subscription price in the United States is $8.00 per annum and outside of the United States for $9.50 per annum.

ASSOCIATION NEWS : VERENIGINGSNUUS

FULL-TIME HOSPITAL AND UNIVERSITY MEDICAL OFFICERS' GROUP

ANNUAL REPORT AND FINANCIAL STATEMENT 1948-1949

The Constitution of this group was approved on 22 September 1948. The group was established for the following reasons:

1. To promote and stimulate the advancement of clinical and research medical institutional practice.

2. To define and establish relationships amongst medical officers in full-time service and between full-time medical staff and hospitals, public and private institutions, Government authorities, the medical profession generally and the public.

3. To promote the professional status and interests of medical officers in full-time employment with particular reference to salaries, promotions, working conditions, vacation and study leave, transfers, pensions, postgraduate medical educational facilities, inter-change appointment with other centres and countries and any other matters.

The membership now stands at 83. The following members were re-elected on 9 September 1949 to serve on the Executive Committee:

Chairman: Mr. S. Kleinot; *Vice-Chairman*: Dr. J. Kaye; *Hon. Sec. and Treasurer*: Dr. V. H. Wilson; *Members*: Dr. J. C. Gilroy, Dr. I. J. Grek, Mr. B. Lewin, Mr. J. G. O. Hamman, Dr. S. Kay, Dr. H. Jackson, Dr. N. Feldman.

During the year five General and 11 Committee meetings have been held. Personal interviews have been conducted with the Director of Hospitals in the Transvaal, the Medical Superintendent of the Johannesburg General Hospital and contact has been made with the Augmented Executive Committee of the M.A.S.A. Throughout, the Group has taken an active part either directly or indirectly in the formation of and improvements in Full-Time Medical Services.

UMI

STATEMENT OF ACCOUNT 1 OCTOBER 1949

Subscriptions Received: £42 11s. 0d. *Total Expenses*: £13 7s. 11d. *Total Assets*: £29 3s. 1d.

Considerable progress has recently been made to improve full-time services and it is sincerely hoped that all members will continue to give their valuable support and that new members will be enrolled to further strengthen the group. Medical practitioners in full-time services wishing for membership are asked to apply to the Hon. Secretary.

V. H. Wilson, M.D., M.R.C.P.

Baragwanath Hospital,
Johannesburg.

THE BENEVOLENT FUND

The following contributions to the Benevolent Fund during March 1950 are gratefully acknowledged:

Votive Cards: *In Memory of*:
Mr. Bergh *by* Dr. J. W. M. Claassens.
Dr. J. M. Smith *by* The Transvaal Chamber of Mines, Dr. Lewis S. Robertson, Dr. A. B. Theron, Mr. P. M. Anderson, The Chairman, Members & Staff of the Silicosis Medical Bureau, The Management & Staff of Union Corporation, Limited, Dr. G. F. Wright, Maandag Tennis Klub, the Royal Johannesburg Golf Club, Mrs. Lintie Becker, Mr. & Mrs. J. Russell, Prof. & Mrs. J. D. A. Krige, Mr. & Mrs. C. Jooste, Dr. & Mrs. S. W. Verster, Dr. & Mrs. B. G. Melle & Famliy, The Ladies' Visiting Committee, Transvaal Memorial Hospital for Children, Dr. James Black, Mrs. M. H. Blakeway and Family, The Matron & Nursing Staff, No. 1 S.A. Military Hospital, Voortrekker Hoogte, Dorothy Geerling, The Secretary for Mines & Staff, Pretoria, Mr. R. E. Grieveson.
Dr. S. K. Montgomery *by* Dr. S. Sieff.
Mrs. I. Bell *by* Dr. G. Stafford Mayer.
Dr. C. F. Beyers *by* Dr. A. van der Poel.
Dr. T. J. R. Scholtz *by* Dr. A. van der Poel.
Dr. T. F. Andrew *by* Dr. A. van der Poel.
J. A. Barron *by* Dr. C. E. L. Burman.
Mrs. Kathleen McGrath *by* Drs. Schwartz & Solomon.
Dr. M. Fine *by* Dr. I. Friedman, Dr. L. J. Jacobson, Mr. I. Norwich, Dr. L. Staz, Ophthalmological Society of S.A., Dr. M. Franks.
Mrs. Lillian Bergh *by* Dr. G. Stafford Mayer.

Total Amount Received from Votive Cards:	£58	0	0
Services Rendered to:			
Matron S. Jewell by Drs. F. H. Dommisse & T. Fichardt.			
Mrs. N. G. H. Cloete *by* Dr. A. W. S. Sichel.			
Mathilda Mendelow *by* Dr. J. Wolfowitz, Drs. Greenwood, Theron & Samuel.			
Mrs. Linton *by* Drs. W. A. Dodds & A. L. Harington.			
Total Amount Received from Services Rendered:	25	15	0
Donations:			
Dr. E. H. Boodrie	1	1	0
Northern Transvaal Branch Members	126	11	7
	£211	7	7

REVIEWS OF BOOKS

ZINC IONS IN EAR, NOSE AND THROAT WORK

Zinc Ions in Ear, Nose and Throat Work. By A. R. Kriel, M.D. (Univ. Dub.), F.R.C.S.I. (Pp. 60. With illustrations). Bristol: John Wright & Sons, Ltd. 1948.

Contents: 1. General Considerations. 2. Clinical Application of Zinc Ionization and Zinc Electrolysis in Chronic Otorrhoea. 3. Zinc Electrolysis, with Special Reference to Zinc Electrolysis for Destroying Tissue in the Nose or Throat.

This a very short book of some 60 pages. Half of it is devoted to general considerations of the physical and chemical facts pertaining to the movement of ions when an electrical current is passed through zinc salt solutions.

The second half deals in a sketchy way with the clinical application of this to sepsis in the ear, nose and throat.

This has long been recognized from a treatment in certain cases of vasomotor rhinitis and chronic otitis media. In the case of the former disease it is fairly pointed out that the treatment does not alter the patient's diathesis and may have to be repeated in hay fever in successive years. The chapter deals with zinc electrolysis used in the nose for various conditions, and also for destroying the tonsils in adults,, which latter would appear a tedious and long drawnout procedure compared with dissection, besides being a doubtful method of complete extirpation of the gland.

BACTERIOLOGICAL TECHNIQUE

Bacteriological Technique. By W. W. W. McEwen, A.I.M.L.T., F.R.M.S. (Pp. 293 + viii. With 70 illustrations. 15s.) London: Messrs. J. & A. Churchill Limited.

Contents: 1. Laboratory Accommodation and Equipment. 2. Care of Laboratory Glassware and Equipment. 3. Preparing Routine Glassware and Apparatus for Sterilization. 4. Special Apparatus for Collection of Specimens. 5. Laboratory Glassblowing. 6. Upkeep of Laboratory Stock. 7. Laboratory Records of Specimens. 8. Laboratory Animals. 9. Animal Post-mortem Technique. 10. Sterility and Sterilization. 11. Centrifuges. 12. Immunity and Immunological Technique. 13. Wassermann, Kahn and Similar Reactions. 14. General Culture Technique. 15. Anaerobic Culture Technique. 16. Stock Cultures. 17. Preparation of Culture Media. 18. Filtration of Media. 19. Culture Media. 20. Bacteriological Filters. 21. Sections. 22. Routine Bacteriological Examination of Water. 23. Routine Bacteriological Examination of Milk. 24. Bacteriological Examination of Shell-Fish 25. Routine Bacteriological Examination of Stools. 26. Routine Bacteriological Examination of Swabs. 27. Routine Bacteriological Examination of Sputa. 28. Examination of Hairs, etc., for Ringworm. 29. Slide Preparations for Microscopical Examination. 30. Stains, Staining Methods and Fixatives. 31. The Microscope.

This very useful little book is described as a *Guide to Medical Laboratory Technicians* and should prove very helpful to those engaged in this type of work. It is most comprehensive, covering practically every aspect of the technical methods employed in an up-to-date laboratory.

Starting off with a description of a well-planned laboratory, Mr. McEwen gives minute details of the care of the laboratory itself as well as of the apparatus. This is an important part of the duties of technicians which it is well to stress .

Chapter by chapter practically all the usual technical methods employed are described—the sterilization of glassware; preparation and sterilization of culture media, including their recipes; staining methods and the making up of staining reagents and so on, including detailed accounts of examinations of water, milk, faeces, etc.

There is a very useful section on the care of animals and the only omission appears to be no account of the actual inoculation of animals such as virulence tests and the biological test for tuberculosis.

A special chapter is included by Eric G. Knowles on the microscope which, especially, is valuable to all medical Technologists.

MEDICINE OF 1949

Medicine of the Year. First Issue (1949). Editorial Direction by John B. Youmans, M.D. (Pp. 143 + v. 35s.) London: J. B. Lippincott Co. 1949.

Contents: 1. Internal Medicine. 2. Obstetrics. 3. Pediatrics. 4. Surgery.

The editor of this book claims that its purpose is to provide the practitioner with a readable, concise presentation of the changing events in medicine during the preceding year and to help him keep abreast of progress. The reviewer agrees that it fulfils this purpose.

In addition to the main branches of Medicine, viz., Internal Medicine, Surgery, Gynaecology, Obstetrics and Pediatrics, the various sub-divisions Allergy, Anaesthetics, Orthopaedics,

etc., are dealt with concisely and satisfactorily by the 21 contributors. At the end of each section a list of up-to-date references is given for those desiring fuller information.

In the section of Internal Medicine, the antibiotics receive due attention. Mention is made of aureomycin in brucellosis, and atypical pneumonia; of chloromycetin in typhoid, and of each agent in the rickettsioses. The pros and cons of streptomycin are evaluated. A sub-section is devoted to the BAL treatment of heavy metal poisoning; the general practitioner should note that satisfactory results have followed its use in the ingestion of arsenic compounds.

The section on Endocrinology is well written. One notes that an effective, purified adrenocortical-stimulating hormone has now been prepared and opens up great diagnostic and therapeutic possibilities. A very practical test for adrenocortical function is described.

The section on Surgery by Warren H. Cole is informative. The intravenous use of fats for nutrition is being developed. A controversy still rages regarding the use of vagus resection for peptic ulcer. Gastric haemorrhage may be controllable by local chemical means. There is a trend towards portocaval shunt for portal hypertension.

It is not possible to review this book more fully in the space allotted, but as a final remark, one is impressed by the emphasis on therapy throughout.

BRITISH SURGICAL PRACTICE

British Surgical Practice, Volume 6. Edited by Sir Ernest Rock Carling, F.R.C.S., F.R.C.P., and Sir James Paterson Ross, K.C.V.O., M.S., F.R.C.S. (Pp. 554 + xxvi, with 303 illustrations. 66s.) Butterworth and Company Limited, 1 Lincoln's Court, Masonic Grove, Durban, 1949.

Contents: 1. Malingering. 2. Manipulative Surgery. 3. Mediastinum. 4 Melaena and Blood in the Stools. 5. Melanoma. 6. Meninges—Meningitis, Acute and Chronic. 7. Mouth and Pharynx, Malignant Disease of. 8. Muscle and Tendon—Diseases and Injuries. 9. Neck—Cellulitis. 10. Neck—Cut Throat. 11. Neck—Cystic Swellings of. 12. Neck—Tuberculosis Glands. 13. Neoplasma—Innocent and Malignant. 14. Nerves—Cranial. 15. Nerves, Peripheral—Injuries. 16. Neuralgia—Trigeminal, Glossopharnygeal. 17. Nose, Nasopharynx and Accessory Sinuses. 18. Odontomes and Epithelial Cysts. 19. Oedema—Traumatic. 20. Oseophagus. 21. Omentum. 22. Optic Nerve. 23. Orbit—Injuries, Infections, Neoplasms. 24. Orthodontics. 25. Orthodontics—Surgery of. 26. Orthoptic Training. 27. Ovary. 28. Oxygen Therapy. 29. Pain—Causalgia. 30. Pancreas. 31. Paralysis—Management of. 32. Parathyroid Gland—Diseases. 33. Pellagra. 34. Pelvic Organs—Displacement. 35. Peptic Ulcer and its Complications. 36. Perforating Ulcer of the Foot. 37. Peritoneum and Peritonitis. Index to Volume 6.

The sixth volume of the series *British Surgical Practice* takes the reader from *Malingering* to *Peritoneum and Pertionitis*. As in the previous volumes, there is subject matter which does not strictly belong to the field of the general surgeon, but which will no doubt prove of great practical value to those practising surgery in the country. This includes conditions such as Malingering, Meningitis, the Optic Nerve, the Orbit, Orthodontics, Orthoptics, Pellagra, etc.

In a chapter on Manipulative Surgery there are illustrated descriptions of the manipulative technique of individual joints and regions, which will prove most useful to those not thoroughly versed in this subject.

Mr. N. R. Barrett contributes an excellent chapter on Lesions of the Mediastinum, and although the subject belongs to the thoracic surgeon, there is much useful information on conditions such as mediastinitis, enlarged mediastinal glands and mediastinal emphysema, which are of importance to the general surgeon.

Malignant disease of the mouth and pharynx is dealt with at great length. Of special interest are the sections on carcinoma of the lip and tongue, conditions which are only too frequently encountered in this country. The relative merits of radiotherapy and surgical treatment are discussed. Radiotherapy is regarded as the treatment of choice, and the indications for surgery are outlined. A detailed description of block dissection of malignant glands in the neck is included.

The chapter on Muscle and Tendon Injuries and Diseases includes a short account of gas gangrene. In addition common lesions such as tenosynovitis, rupture of muscles and severed tendons are dealt with in detail, with excellent descriptions of the technique of operative repair. This is a chapter which should be carefully studied by everyone who has to deal with traumatic surgery.

In two short chapters on Cellulitis of the Neck and Cut Throat the details of treatment are emphasized—excellent references for those who are suddenly called upon to deal with these conditions.

Francis d'Abreau contributes a very interesting chapter on Cystic Swellings of the Neck, dealing with sublingual dermoid, thyroglossal cyst, branchial cyst and cystic hygroma.

Tuberculous glands in the neck are discussed at length. The author stresses the value of constitutional treatment and emphasizes that all surgery can accomplish is to assist nature to overcome the infection.

Peripheral Nerve Injuries are dealt with at great length, with detailed descriptions of exposures and operative technique. This is yet another chapter which will prove invaluable to every surgeon dealing with traumatic lesions.

In the chapter on the Nose, Nasopharynx and Accessory Sinuses common lesions such as Allergic Rhinitis, Sinusitis and Adenoids are considered in detail, while less common lesions which belong to the Ear, Nose and Throat Surgeon are also adequately dealt with.

P. R. Allison contributes an excellent chapter on Diseases of the Oesophagus. He deals particularly with carcinoma of the oesophagus, and includes excellent descriptions of resection of the upper, middle and lower thirds of the organ.

The account on Causalgia by Professor Boyd is interesting, but one feels that the author is rather too optimistic about the excellent results of sympathetic denervation in this troublesome condition.

Professor Illingworth deals with Diseases of the Pancreas and gives a good account of the operation for the excision of the organ.

The outstanding chapter in the volume is no doubt that on Peptic Ulceration by Sir Heneage Ogilvie. This is a complete dissertation which in itself is an excellent monograph on the subject. The author deals with all aspects of peptic ulceration and stresses in particular surgical treatment. The indications for surgery are discussed, pre- and post-operative care are described and operative technique is dealt with in some detail.

The last chapter, namely that on the Peritoneum and Peritonitis, is rather brief, but it nevertheless covers adequately the condition of acute diffuse peritonitis and its treatment.

As in previous volumes there has been a careful selection of authors, all of whom are experts in their particular subject. The general high standard of the series as a whole is thus maintained, and there is no doubt that the complete series will prove one of the best reference libraries that any surgeon could wish to have on his bookshelf.

SOCIAL MEDICINE

Recent Advances in Social Medicine. By Alan Carruth Stevenson, B.Sc., M.D.Glas., M.R.C.P.Lond., D.P.H. (Pp. 241, with illustrations and tables. 18s.) London: J. & A. Churchill Ltd. 1950.

Contents: 1. Measurement of Growth in Children. 2. Infant Mortality (1). 3. Infant Mortality (2). 4. The Health of Children in Communal Day Nurseries. 5. The Unmarried Mother and her Child. 6. Problem Families. 7 School Medical Inspection. 8. Some Applications of Statistical Methods. 9. Notes on Three Subjects of Interest: Psychosomatic Illness and Social Medicine. The Adolescent in Industry. 'Pulheems.' Index.

The publication of a volume entitled *Recent Advances in Social Medicine* is an indication of the increasing use of the term Social Medicine. The author prefers to leave discussion of the meaning of the term to others. His choice of subjects, however, is described as concerning 'matters which are dealt with in teaching undergraduate and post-graduate students in all University departments of preventive medicine'. The clinical practice of Social Medicine in general or specialty practice receives little direct discussion. In the light of other British writings on the subject of Social Medicine, it appears that there is an increasing tendency to substitute the use

of this term for those of public health and preventive medicine. It would seem that there is considerable scope for discussion and review of the meaning of the word 'Social' when used in the descriptive term Social Medicine.

While the present volume does not add to our understanding of the functions and meaning of Social Medicine, it contains a series of articles, each of which throws considerable light on the particular subject discussed. The chapter on the measurement of growth in children is a very able review of the various indices which have been used for assessment of growth and health. It includes a selected bibliography on the subject of growth studies which will prove of inestimable value to both the student and practitioner.

In his essay on School Medical Inspection the author discusses the value of the routine inspection of schoolchildren. His objective critical approach to a system which is now over 40 years old and is well established in British schools is most refreshing. He says of it: 'It is surprising how little the system has been submitted to criticism and evaluation in the medical literature. Indeed, the contrast between private and public opinions on the matter is remarkable'; and concluding his clear and provocative review of the existing service he remarks: 'Nothing could be more disastrous to sustained interest and clinical standards than a surfeit of school inspections.' To those interested in the periodic health examination, reading this chapter will be a most stimulating experience. While some may disagree with the author's conclusions, few will deny that he has exposed the defects intrinsic in a rapid physical inspection as a means of assessing health.

Each subject dealt with receives the same stimulating consideration. The author has a gift of expression becoming all too uncommon in modern medical writing, and the standard of publication is of the high order which we have come to associate with publishers such as Messrs. J. & A. Churchill. Minor discrepancies between references in the text and those in the bibliography are noted and if, as is hoped, a second edition of this volume is produced, these should be corrected.

ALLERGY: ITS ABC

Allergy: What it is and What to do about it. By Harry Swartz, M.D. (Pp. 210 + xiv. 9s. 6d.) London: Victor Gollancz Ltd. 1950.

Contents: *Foreword. Introduction.* 1. How old is Allergy? 2. The Discovery. 3. Coining the word. 4. Man and Animal. 5. Atopic and Non-Atopic Allergy. 6. Drugs, Oils and Infective Agents. 7. Physical Agents: Heat, Cold, Light. 8 Histamine or the 'H' Substance. 9. Personality Patterns. 10. Allergy and Disease in General. 11. The Newer Idea. 12. The Common Ways of Allergy. 13. The less Common Ways of Allergy. 14. What can be done about Allergy? 15. Drugs and Aids. 16. 'Cures'. 17. Industry and Allergy. 18. Control. Glossary. Index.

In this short book, interestingly written and easy to read, the layman will find answers to most if not all of the problems and mysteries concerning the subject of allergy. To-day, when almost every parent is told by his medical practitioner that one or more of his family is suffering from allergy in one of its numerous forms, it would be useful to refer those of them who are interested in acquiring further knowledge to a book such as this.

The chapters on the history and discovery of allergy and on the allergies acquired through various occupations will prove of interest to the general reader, while the section dealing with the latest developments in treatment may be of use to the general practiioner.

AETIOLOGY OF MURDER

The Show of Violence. By Fredric Wertham, M.D. (Pp. 279. 15s.) London: Victor Gollancz Limited. 1949.

Contents: 1. Psychiatry in the Courtroom. 2. The Shadow of Madness. 3. How to Commit a Murder in New York. 4. Masks have no Ears. 5. The Murderer I Never Saw. 6. Manhattan Tragedy. 7. Disorderly Conduct. 8. Medea in Modern Dress. 9. Mathematics of Murder.

All those interested in the problems of the killer will be entertained as well as fascinated by this extremely readable account of a study of the motives for murder. Dr. Wertham has had a very considerable medico-legal experience as a leading American psychiatrist in this field, and he presents some six cases which are of great and compelling interest, whether one regards them as psychopathic or psychiatric problems on the one hand or, on the other hand, as half a dozen stories of distorted lives each carrying its own tragic and dramatic tale.

The important point which Dr. Wertham makes is that, properly handled, the psychopath or the victim of a psychopathic personality (i.e. a person with an abnormal mental make-up) can be prevented from perpetrating this type of murder. The book is also a lively indictment of the many psychiatrists who disgrace their calling by the type of evidence they are prepared to give in the Courts. The author inclines to the view that the concept of the 'irresistible impulse' is one of unmitigated metaphysics. He himself believes that all compulsions are innocent in nature.

His fundamental approach to the problems of disorderly behaviour resulting in murder is an extremely commonsensical one, and the statistics he quotes in the chapter entitled *Mathematics of Murder* are certainly very arresting as well as alarming. He makes the point that there is no lack of material to study the experiment of murder, since, in the United States, a murder is committed every 45 minutes. 'There are times and places where it outranks even tuberculosis' as a cause of death (p. 243).

His opinion about unsolved murders is also striking. Apparently a murderer commits a perfect crime in the United States every two hours, 'which is considered oftener than a golfer makes a hole in one' (!). Dr. Wertham's style is lively and entertaining and readers will be amused by the little verse he quotes when describing the picture presented by two psychiatric experts who were engaged by the defence and also authorized by the Court:

> Physicians of the utmost fame
> Were called in turn, and when they came
> They answered as they took their fees,
> There is no cure for this disease.

The extraordinary pass to which affairs have come in the United States certainly lends much point to the author's suggestion that 'instead of spending so much of the taxpayers' money on the medico-legal responsibility of individual culprits, it would be better to study the sense of responsibility of the experts. It is not easy to draw the line where bad psychiatry ends and bad faith begins.'

BLOOD AND MARROW ATLAS

An Atlas of the Blood and Bone Marrow. By R. Philip Custer, M.D. (Pp. 321, with 285 figures, 42 in colour. £6 7s. 6d.) Philadelphia and London: W. B. Saunders Company. 1949.

Contents: Part I. The Hemolytopoietic System: 1. Terminology. 2. Hematopoiesis. 3. Normal Blood and Bone Marrow. Part II: Disorders of the Blood and Bone Marrow: 4. Classification. 5. Deficiency Anemias. 6. Aplastic and Hypoplastic Anemias. 7. Displacement of Bone Marrow. 8. Hypersplenism. 9. Hemolytic Anemias. 10. Ill-Defined Anemias. 11. Hemorrhagic States. 12. Effects of Physical and Chemical Agents. 13. Leukocytosis, Leukemoid Reactions, and Leukopenia. 14. Infections. 15. The Leukemias. 16. Polycythemia. 17. Technic. Index.

This book is planned for the use of the clinician, the clinical haematologist and the pathologist. The author ably emphasizes the importance of regarding the peripheral blood and bone marrow reaction pictures not just as blood diseases, but as reaction to and part of general disease.

The outsanding feature of the Atlas is the very large number of photomicrographs of stained and wet smears of peripheral blood, bone marrow smears and bone marrow sections. There is a total of 288 figures, of which some 40 are in colour. Many of the coloured illustrations of marrow smears and perpiheral blood smears are magnified 2.280 times. To those who are accustomed to these high magnifications the Atlas is of importance; whilst those unaccustomed to such high magnifications have a new world of haematological morphology opened up to them. There are enough coloured illustrations to include most types of cell, normal and abnormal. Black-and-white photomicrographs have a value limited for the very reason that they are not in colour,

but nevertheless they do show accurately cell distribution in marrow and blood, the legends to the figures and the lettering and pointers in the photomicrographs being sufficiently descriptive to indicate the morphological character of the cells.

The book covers the whole field of haematological disorders and includes a well-illustrated section on virus, rickettsial, spirochaetal, bacterial and other infectious diseases.

The author's classification is no better and no worse than any other accepted classification. The terminology as far as the white cell and the platelet series are concerned, is that recommended by the Joint Committee of the American Society of Clinical Pathologists and the American Medical Association. The author has, however, not adopted the red cell terminology recommended by the committee in which 'rubricyte' displaces 'erythrocyte', and the 'rubriblast' the 'erythroblast'. The terminology used is that which is most generally accepted in the English-speaking world to-day.

The author maintains a broad outlook throughout the book and warns against undue reliance being placed in diagnosis on the marrow and peripheral blood picture, to the exclusion of other clinical and laboratory investigations.

References to treatment are of a genral but nevertheless comprehensive nature. The section on normal blood and bone marrow covers very adequately the features of these tissues at all ages.

The chapter on technique includes the description of methods of obtaining bone marrow smears and sections and peripheral blood smears. The author emphasizes the importance of good technique in making marrow smears in particular and illustrates for comparison smears made by good and by bad techniques.

The book is a valuable contribution to haematology and will undoubtedly be stimulating to those who wish to advance their knowledge of clinical haematology.

PREGNANCY DIAGNOSIS

Pregnancy Diagnosis Tests: A Review. Commonwealth Agricultural Bureaux Joint Publication No. 13. By Alfred T. Cowie, B.Sc., M.R.C.V.S., Ph.D. (Pp. 283. 15s.) England: Commonwealth Agricultural Bureaux. 1948.

Contents: 1. Introduction. 2. Clinical Methods of Pregnancy Diagnosis. 3. Tests Based on Hormonal Investigations of Body Fluids. A. Gonadotrophic Hormones. 4. Tests Based on Hormonal Investigations of Body Fluids. B. Oestrogenic Hormones. 5. Tests Based on Hormonal Investigations of Body Fluids. D. Other Hormones. 7. Reviews. 8. Tests Based on Enzymic Investigations of Body Fluids. 9. Tests Based on other Biochemical Investigations of Body Fluids and Tissues. 10. Tests Based on Various Physiological Phenomena. 11. Tests Based on Immunological Phenomena. 12. Tests Based on Physical Investigations of Body Fluids and Tissues. 13. Summary and Conclusions. 14. Bibliography. 15. Index to Tests. 16. Animal Index.

This publication is a fairly comprehensive review of tests employed for the diagnosis of pregnancy. The author places the tests in seven categories, viz., the tests based on clinical methods; tests based on hormone, enzyme, biochemical or physical investigations of body fluids; tests based on physiological or immunological phenomena. From the practical point of view this is probably as good a way to group the great variety of tests in use as any.

Dr. Cowie's review aims at being more than an annotated bibliography. Because of the broad comparative approach there is a considerable amount of information about the use of tests to detect pregnancy in other animals besides man.

It is unfortunate that the author failed to note that the first paper recording the use of *Xenopus* as a test animal for pregnancy diagnosis in man was published by Shapiro and Zwarenstein as early as 1933 (18 October, Proc. Roy. Soc. S. Af.), thus ante-dating Bellerby's communication (1934) very considerably.

Dr. Cowie has performed a very useful task in gathering together such a very considerable amount of information within the compass of a single, manageable volume which will be of great value to all those interested in the theory and the practice of pregnancy diagnosis.

A REVIEWER REGRETS

A Doctor Regrets. . . . Being the First Part of 'A Publisher Presents Himself'. By Donald McI. Johnson. (Pp. 242, with 64 illustrations. 12s. 6d.) London: Christopher Johnson Publishers Ltd. 1949.

Contents: 1. Lancashire. 2. Cheltenham. 3. Cambridge. 4. London. 5. East Greenland. 6. Hospitals. 7. Labrador—1. 8. Labrador—2. 9. General Practice—1. 10. General Practice—2. 11. Wavertree. 12. A Doctor Regrets.

This volume is an autobiographical account of the author's medical career. It is not clear to the reviewer what necessity required its publication. The author appears to have been discontented with the career he chose as a medical practitioner, he qualified for but was unable to undertake practice at the Bar, his entry into politics was apparently also unsatisfactory and his present occupation appears to be that of a publisher with the object of presenting 'high-class books of informative and thoughtful nature'.

The volume under review is the first part of 'A Publisher Presents Himself' and this book is, therefore, presumably an example of a 'high-class book of informative and thoughtful nature; (displaying the need for) intelligence rather than intellectualism, understanding as well as knowledge'.

Although the author took part in a scientific expedition to Greenland and was afterwards a medical practitioner in Labrador, his record of his career as a medical practitioner is extremely stale, very uninteresting and pedestrian. One fact, however, emerges clearly. The book reads more like a record of a mere existence rather than of an exciting life. Another fact which emerges equally clearly is that the author loved his father and his mother.

MATERIA MEDICA

Bennett's Materia Medica and Pharmacy for Medical Students. By Reginald R. Bennett, B.Sc. (Lond.), F.R.I.C. Revised by H. G. Rolfe, B.Sc. (Lond.), F.R.I.C. (Pp. 276+xxviii. 16s.) London: H. K. Lewis and Company Limited. 1950.

Contents: 1. General Remedies. 2. Local Remedies. 3. Extraneous Remedies. Dose Tables. Appendix.

This book provides a concise account of preparations in the British Pharmacopoeia 1948, and deals mainly with the sources and characters of drugs. However, there is extremely inadequate information about the actions and uses of drugs, at the most a few words, and in many instances, e.g. digitalis, deoxycortone, emetine, barbiturates, penicillin, just about nothing at all.

Another defect, since this book only mentions B.P. preparations, is that important drugs, e.g. streptomycin, proguanil, propylthiouracil, are not considered in a book dated 1950.

Toxic features of such important remedies as sulphonamides, arsenic, lead, opium alkaloids, are not indicated.

Those interested in the origin and pharmaceutical aspects of official preparations will find this book useful, but it lacks much that medical students need to know.

SOCIETY AND ITS CRIMINALS

Society and its Criminals. By Paul Reiwald. (Pp. 315 + xix. 21s.) London: William Heinemann Limited.

Contents: 1. The Social and the Asocial Person. 2. The Resistance of the Lawyer to Psychology. 3. The Judge and the Father. 4. The Criminal in Phantasy: Part I. 5. The Criminal in Phantasy: Part II. 6. The Criminal of Reality. 7. The Contrary Ones. 8. The Genesis of Punishment. 9. Aggression in the Criminal Law. 10. The Future of Criminal Law.

This book offers a new approach to criminology. Freud is the prophet; the author his disciple. Armed with the doctrine of the prophet, the disciple sallies forth to overthrow long-established beliefs and faiths. Believing that with the help of psycho-analysis we can move from the conscious to the unconscious and thereby come closer to an understanding of the criminal and our relationship to him, this book makes

a serious attempt to psycho-analyse 'society and its criminals'. Unless the reader approaches this book with an open, enquiring mind, he is not likely to proceed beyond the first chapter, for this new treatment of an age-old problem requires a willingness on the part of the reader to reconsider and revalue firmly-held beliefs and opinions. If this condition is fulfilled, he will find much to stimulate his thoughts, even if at times he may feel incredulous, irritated or frankly baffled.

The main thesis of the book, presented with considerable learning and documentation, is that 'mankind has the criminal which it desires, and it desires him for the satisfaction of psychic needs' or, as the author states it elsewhere, 'the criminal is necessary to man . . . He needs him for emotional reasons. Consequently, he organizes the fight against him so that at the same time it serves the purpose of maintaining crime'. So startling a proposition will be received with considerable reserve and scepticism. Nevertheless, the author succeeds in giving very plausible support to his thesis by a searching enquiry into the reasons for the public's undeniable demand for murders—whether in the form or detective stories, crime films or actual court trials. These chapters form the most interesting in the book and leave one with the rather uncomfortable conviction that society has some psychic need for crime, whatever form it may take.

In the final chapter the author makes an appeal for the establishments of this basic principle in our relationships with the criminal: 'treatment, instead of reacting emotionally'.

To all those who are concerned with the modern problem of crime or whose work brings them into contact with criminal law, this book can be recommended for serious reading.

BEDSIDE DIAGNOSIS

Bedside Diagnosis. By Charles Mackay Seward, M.D., F.R.C.P. (Edin.). (Pp. 372 + xii. Illustrated. 17s. 6d.) Edinburgh: E. & S. Livingstone, Ltd. 1949.

Contents: 1. Psychogenic Symptoms. 2. Some General Considerations Regarding Pain. 3. Head Pain. 4. Thoracic Pain. 5. Epigastric Pain. 6. Umbilical Pain. 7. Hypogastric Pain. 8. Lateral Abdominal Pain. 9. Anaemia. 10. Epistaxis. 11. Haematemesis. 12. Haematuria. 13. Haemoptysis. 14. Cough. 15. Dyspnoea. 16. Tachicardia. 17. Disphagia. 18. Vomiting 19. Diarrhoea. 20. Jaundice. 21. Debility and Loss of Weight. 22. Pyrexia. Index.

Dr. Seward has written this book as a guide to the student in the process of diagnosis from the symptoms and signs actually presented by the patient in clinical practice. Dr. Seward discusses the main presenting symptoms of disease and deals with each in a standardized method, beginning with a synopsis of the causes of the symptom, the physiology of the symptom and then the diagnostic approach. Finally the diseases mentioned are briefly described, but as each is described once only there is necessarily considerable cross reference between different sections of the book.

Such a lay-out has obvious advantages, but it has the serious defect that the common and dangerous conditions that may produce the symptom are not necessarily considered first, as they should be in clinical practice, but may be described anywhere in the chapter on that symptom or may even be described in another section. Thus, tuberculosis, the most important cause of haemoptysis, is not described prominently and at the head of this chapter, as its importance merits; but the reader is referred to another section dealing with pyrexia for its description. Nowhere is the supreme importance of an X-ray investigation of the lungs in haemoptysis emphasized.

Another fault of this book is that Dr. Seward has attempted too much in too small a compass. For this reason descriptions of diseases are often too short to be useful in identifying the condition and often no attempt is made to evaluate the importance of laboratory or X-ray investigations. Also the language used often suffers from this compression. Thus, dealing with the subarachnoid haemorrhage, it is stated 'a lumbar puncture is bloodstained and is diagnostic', a statement which may actually be misleading as no mention is made of the importance of discolouration of the spinal fluid on standing.

In spite of these faults, however, Dr. Seward's book will undoubtedly prove useful to students in the wards as a handy and portable guide in clinical diagnosis.

YEAR BOOK OF MEDICINE

The 1949 Year Book of Medicine (July 1948-May 1949). Edited by Paul B. Beeson, M.D., J. Burns Amberson, M.D., George R. Minot, M.D., S.D., F.R.C.P., William B. Castle, M.D., S.M. (Hon.), Yale, M.D. (Hon.), Utrecht., Tinsley R. Harrison, M.D., George B. Eusterman, M.D. (Pp. 831. With illustrations. $4.50.) Chicago: The Year Book Publishers Inc. 1949.

Contents: 1. Infections. 2. The Chest. 3. The Blood and Blood-Forming Organs and the Kidney. 4. The Heart and Blood Vessels. 5. The Digestive System.

Part I on infections deals mainly with the therapeutic value of aureomycin and other aspects of therapy.

In the next section there is naturally a large portion devoted to tuberculosis, particularly to the active and surgical therapeutic aspect.

The Editors have abstracted many articles on the advantages of iliac puncture over sternal marrow puncture in Part III. Lower nephron nephrosis also features prominently.

One is surprised (in Part IV) that there are practically no abstracts on the V leads in the electrocardiography section, but editor Harrison explains this in his opening paragraph. Tetraethyl-ammonium chloride and the peripheral vascular disease literature have been well abstracted. Anticoagulants, of course, occupy several pages.

Only on reading the last section does one realize how much has been written recently on the emotional factors in gastro-intestinal disease. Pancreatitis is almost as well represented as liver disease and liver function.

To those not familiar with the *Year Book*, suffice it to say that here lies the answer in keeping abreast of the current, unwieldy medical literature. Each abstract is full enough to give the main content of the relevant article, while the excellent subject and author index makes it a ready reference.

ORIGIN OF MEDICAL TERMS

The Origin of Medical Terms. By H. A. Skinner, M.B., F.R.C.S. (C.). (Pp. 379 + viii. 54s.) London: Baillière, Tindall & Cox. 1949.

Dr. Skinner has prepared an interesting dictionary of the origin of medical terms. The increasing interest in the history of medicine should make this modest contribution of value to those who seek a medical hobby.

An interest in medical terminology will undoubtedly do much to clarify and improve current usage.

The volume is pleasantly designed, and a useful bibliography provides the enquiring reader with an excellent guide for further study.

CORRESPONDENCE

PART-TIME POSTS IN HOSPITALS

To the Editor: A meeting of the Southern Transvaal Branch was held recently to consider the working of the 'Free Hospitalization' scheme.

At this meeting there was much talk—with strong justification—about the breach of faith on the part of the Transvaal Provincial Administration.

But there is a breach of faith on the part of certain medical men, to which I feel the greatest publicity should be given, as it may be used against the profession with telling effect.

I refer to the fact that certain practitioners—usually senior men—have accepted posts which require the giving of a certain number of hours' service, and that they are not putting in the time which they have contracted to do.

This breach of faith is particularly deplorable, as by holding posts to which they find they cannot devote the contracted time, they are keeping less busy practitioners out of these posts.

I feel this matter is of sufficient importance to merit your editorial comment.

'*Equity*.'

8 April 1950.